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IMPERIAL MYCOLOGICAL INSTITUTE
3 JAN 1940

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TROPICAL DISEASES BULLETIN.

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TROPICAL OPHTHALMOLOGY.

A Review of Recent Articles. XXXIV.*

Trachoma.—Some of the problems presented by the disease have been discussed by Taborisky[1]. This observer has had an exceptional experience of trachoma in Palestine, both amongst the school-children and the general population. He admits that competent ophthalmologists might disagree regarding the diagnosis of any particular case of trachoma in the early stage; the progressive development of a deep conjunctival inflammation (hyperaemia, thickening and papillary hypertrophy) followed later by the formation of follicles is, however, characteristic. In heavily infected areas the virus on occasion may reach the eye in such small quantity or with such attenuated virulence as to produce no reaction; more often in such cases it may induce an inflammation which heals spontaneously or becomes chronic. A relative immunity may occasionally be present, and, should this be overcome, a true trachoma, though slight or atypical, may develop.

Burnet, Cuénod and Nataf[2] have noted how closely the cell inclusions found in trachoma resemble those seen in lymphogranuloma and suggest that those drugs which are useful in the experimental form of the latter disease might prove equally serviceable in the case of the former. They have therefore employed diaminodiphenylsulphone in the treatment of trachoma and have recorded their results. These appear to be fairly promising and the authors consider they have made a step forward in the treatment of the disease. Chang[3] has treated eighty cases of trachoma with ten per cent. solution of quinine sulphate according to the method described by Selinger and has formed a favourable opinion of its efficacy. The solution is rubbed for one minute into the conjunctiva by means of a cotton applicator. The treatment is carried out twice weekly at first and the sittings

* For the 31st of this series see Vol. 36, pp. 455–458.

[1] Taborisky (J.). Sur les problèmes du trachome et les méthodes de leur solution.—*Rev. Internat. du Trachome.* 1939. Apr. Vol. 16. No. 2. pp. 65–79. [20 refs.]

[2] Burnet (Et.) Cuénod (E.) & Nataf (R.). Essai de chimiothérapie du trachome par un dérivé glucosé du "4 : 4' diaminodiphénylsulfone"; efficacité sur le pannus trachomateux.—*Arch. Inst. Pasteur de Tunis.* 1939. Mar. Vol. 28. No. 1. pp. 11–28.

[3] Chang (S. P.). Clinical Experiences with Quinine Treatment of Trachoma.—*Chinese Med. Jl.* 1939. May. Vol. 55. No. 5. pp. 439–447.

gradually reduced later. Some patients were found hypersensitive to the quinine and these developed an eczematous condition of the lids which subsided on ceasing the treatment.

STEWART[4] has come to the conclusion that trachoma is caused by a virus whose most conspicuous form is the Prowazek-Halberstaedter inclusion body. A free extra-cellular stage (the initial body) also exists; this resembles a pleomorphic bacterium. The initial body enters the cytoplasm of an epithelial cell and increases in size; several such may coalesce and form the kidney-shaped mass known as the Prowazek-Halberstaedter body. Elementary granules which stain red with Giemsa subsequently appear in this body. Initial bodies, both extra-cellular and intra-cellular, stain a dark blue with Giemsa. Filtration through collodion membranes impermeable to bacteria is uncertain, but the virus can be concentrated on the upper surface of such membranes. Baboons and grivet monkeys are susceptible to infection. The virus is killed by drying and infected cloths are dangerous for a short time only. The rôle played by lice in spreading the disease requires further investigation.

Assuming trachoma to be a Rickettsia infection POSTIC[5] has continued the researches of DERKAC on the presence of the Weil-Felix reaction in trachoma. He obtained a positive reaction in 68 per cent. of trachomatous patients against a positive rate of 32 per cent. in non-trachomatous eye patients. One hundred and twenty cases of trachoma were tested and twenty-five controls. TRAPESONTZEWA[6] believes that the Rickettsia found in the gut of lice inoculated with trachomatous material have no connexion with trachoma but are merely incidental. POLEFF[7] claims to have successfully cultivated trachoma Rickettsia *in vitro* by using tissue culture. Human placenta was found a suitable tissue, but he suggests that corneal tissue obtained from a foetus or a recently deceased person might prove even more effective. Thin shavings of infected conjunctiva must be used for implantation, mere scrapings are insufficient. The beneficial effect on the disease of an absence of such deleterious factors as scorching sun, heat, smoke, and dust-laden winds has been noted by MOTAIS[8]. He had charge of 5,000 Tonkingese coolies working on rubber plantations in Cambodia and Cochin China and found that only 0·5 per cent. showed signs of trachoma, whereas in Tonking, their native country, from 50 to 80 per cent. of the population is stated to be affected. The coolies were, of course, medically inspected before recruitment, but even so the difference is striking and Motais believes that the favourable climatic conditions may be largely responsible.

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[4] STEWART (F. H.). The Aetiology of Trachoma.—*Brit. Jl. Ophthalm.* 1939. June. Vol. 23. No. 6. pp. 373–380. With 5 figs. [12 refs.]

[5] POSTIC (Svetozar). L'importance de la réaction Weil-Félix dans le trachome, ainsi que l'apport de l'étiologie Rickettsienne du trachome.—*Rev. Internat. du Trachome.* 1939. Jan. Vol. 16. No. 1. pp. 31–39.

[6] TRAPESONTZEWA (C.). A propos des "Rickettsia" du trachome.—*Rev Internat. du Trachome.* 1939. Jan. Vol. 16. No. 1. pp. 40–47.

[7] POLEFF (L.). Passage des "corpuscules Rickettsoides" du trachome sur le tissu de l'oeil humain hors de l'organisme et quelques observations générales sur la culture des dites formations in vitro.—*Rev. Internat. du Trachome.* 1939. Apr. Vol. 16. No. 2. pp. 79–87. [12 refs.]

[8] MOTAIS (F.). Un changement de terrain et de conditions d'existence, peut-il atténuer la virulence de l'endémie trachomateuse ?—*Rev. Internat. du Trachome.* 1939. Apr. Vol. 16. No. 2. pp. 88–92.

Glaucoma.—Some points concerning *Epidemic Dropsy Glaucoma* have been recorded by KIRWAN[9]. The disease is painless and all the more dangerous in consequence; it usually occurs in the later stages of epidemic dropsy, but may be accompanied by only mild signs of the constitutional disease if the intra-ocular capillary epithelium is specially susceptible to the toxin. Dietetic and medicinal treatment is unsuccessful and eserine is actually dangerous. Operative treatment is unnecessary as long as the visual fields remain unimpaired, but it should be immediately adopted when field defects appear. A modification of Lagrange's operation is recommended as Elliot's operation, though temporarily successful was often found liable to be followed by late complications. BHADURI[10] advocates early recognition of the disease and stresses the importance of haloes as a warning symptom. He has found pilocarpine in two per cent. and four per cent. solution useful and has adopted Lagrange's operation with encouraging results.

Cataract.—A review of the intracapsular operation for cataract has been made by BUXTON[11]. The opinions of many authorities are quoted by the author who himself had considerable experience at the Shikapur clinic. He prefers the classical operation and employs a corneo-scleral suture, capsulotomy with Vogt's capsule forceps, anterior chamber irrigation, and peripheral iridectomy. He concludes that though the intracapsular operation may possess some advantages it involves an increased risk of certain complications. It may, however, be preferable for selected cases in such countries as India, where patients, who require it, are unlikely to return for a capsular discission.

The *Bulletin of the Ophthalmological Society of Egypt*[12] for the year 1938 records the proceedings of the Thirty-fifth Session of the Society. The subject of retinal detachment led to an active discussion in which many members took part. Dr. Max MEYERHOF described some cases of sellar tumour which appeared to benefit from X-ray treatment, but he expressed the opinion that operation is the only sure therapeutic measure in the majority of the cases. Dr. M. M. BAKRY claimed some striking successes in the treatment of optic atrophy by retrobulbar injections of atropin combined with measures taken to reduce the intraocular pressure together with suitable constitutional treatment.

H. Kirkpatrick.

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[9] KIRWAN (E. O'G.). The Treatment of Epidemic Dropsy Glaucoma.—*Trans. Med. College Re-Union.* Calcutta. 1938–39. Vol. 2. pp. 179–182.

[10] BHADURI (B. N.). Some Observations on Epidemic Dropsy Glaucoma.—*Trans. Med. College Re-Union.* Calcutta. 1938–39. Vol. 2. pp. 186–188.

[11] BUXTON (Robert). Intracapsular Extraction of Cataract with Forceps.—*Brit. Jl. Ophthalm.* 1939. Aug. Vol. 23. No. 8. pp. 505–538. [61 refs.]

[12] BULLETIN OF THE OPHTHALMOLOGICAL SOCIETY OF EGYPT. 1938. Vol. 31. Session 35. pp. xxxvi + 118. With numerous illustrations.

PLAGUE.

PRÉCIS OF ABSTRACTS IN THIS SECTION.

YANG *et al.* (p. 960) describe how, in Fukien, plague spread from the ports along the waterways to the interior. Small epidemics occurred year after year to diminish after a decade, but sporadic cases were found in the interepidemic periods. The sanitary condition of many

Chinese towns is bad and rat infestation constant. Fumigation and poison bait are used and for shops the division of rat-proofing measures to protect the three categories, bulk stock, replenishment stock and retail goods proved economically successful. The military authorities co-operated with the civil in an excellent organization.

Rao (p. 962) believes that plague is endemic, though frequently not recognized, in Calcutta and suggests a systematic examination of the rodent population and an estimation of its relative immunity.

Sussini (p. 962) traces the history of plague in S. America. Anti-plague measures in the ports have been conducted on well-known lines and in some instances have reduced the rat population by 70 per cent. Alfarado and De la Barrera (p. 962) report that, in the Argentine, sylvatic plague epizootics appear in winter and fade out in spring. Human infection is not common, but may take the pneumonic form.

Castellani (p. 963) describes a medium containing glycerol and rhamnose for the differentiation of *P. pestis* from *P. pseudotuberculosis rodentium.* Rao (p. 963) shows that the plague bacillus requires certain amino-acids for growth, but accessory growth factors are not essential.

Tumansky (p. 964) shows that *Proteus vulgaris* is antagonistic to *P. pestis,* and this may account for the rapid disappearance of the latter in decomposing corpses, but plague bacilli can remain alive and virulent in rat or guineapig cadavers for 10 months or even longer in bone marrow uninfected by Proteus. Russo (p. 964) shows that virulence was not modified and considers that acari, which live on mummified rats, may become infected.

Breininger (p. 964) relates the action of calcium chloride on *P. pestis* under various conditions. Favorissova (p. 965) describes the effect of phage on cultures of *P. pestis.*

Russo (p. 965) states that flesh eating flies of the genus Sarcophaga, in all stages of development, offer favourable conditions to the multiplication and diffusion of plague bacilli.

In Kenya, Roberts (p. 965) shows that the most important factor in the incidence of plague is the density of the *R. rattus* population. He (p. 965) reports that a high mortality in *field* rodents in Kenya was due, not to plague, but to Rift Valley fever. In the absence of proof it appears wrong to attribute outbreaks of plague in man to field rodents and their fleas.

van Campenhout (p. 966) states that *Mastomys ugandae* is a carrier of sylvatic plague in the Congo, but *R. rattus* plays no part.

Lobanov and Fedorov (p. 966) note a distinct seasonal difference in the reaction of gerbils to plague infection. Korobkova *et al.* (p. 967) conclude that agglutination reactions in spermophils are not likely to be valuable in determining the epidemiology of plague.

In Western Canada Gibbons (p. 967) reports that no plague infection was found in 3,569 wild rodents and 7,582 rodent fleas examined. No infection with *Rickettsia dermacentroxenus* was found in a large number of ticks, but several were infected with *Bact. tularense.*

Jellison (p. 968) shows that predatory species of birds may transport flea infested rodents and may serve as accidental hosts of rodent fleas.

Eskey and Haas (p. 968) show that the fleas of wild rodents and of domestic rats are equally capable of becoming infected. They may harbour virulent plague bacilli in the gastrointestinal tract for as long as 130 days before becoming infective. For determining plague in

wild rodents the most effective method is the inoculation of the bodies of captured fleas into guineapigs. The fleas should be killed by HCN and transmitted to the laboratory in 2 per cent. salt solution. DEVIGNAT (p. 969) uses Broquet's medium (calcium carbonate 2, glycerin 20, distilled water 80) for clearing fleas for examination. This prevents putrefaction but ensures the survival of the bacilli up to six days so that they may be isolated for bacteriological examination.

MURDOCK (p. 969) records an outbreak of pneumonic plague in nurses, attendants and doctors following the admission of a patient to hospital. Of 16 affected, 15 died.

In a child described by FARINAUD (p. 970) streptococci, and later Gram-negative bacilli, were found in buboes. Inoculated guineapigs died of acute plague, and the streptococci were of low virulence.

Mice which ingested M & B 693 to the amount of about 2 mgm. per gm. weight daily, and which were inoculated with 10,000 lethal doses on the 5th or 6th day, ingestion of the drug being continued thereafter, survived. DURAND (p. 970) reported that larger infecting doses often proved fatal, but even then the bacilli could not be recovered from the dead animals. Most of the surviving animals showed considerable immunity. SCHÜTZE (p. 971) shows that white mice are less resistant to plague than white rats. M & B 693, soluseptasine and sulphone were given, before or with the infection, to these animals and it was found that M & B 693 was the most successful. Serum prepared against the Otten living vaccine was about equal to M & B 693 in prophylaxis and the author suggests that a combination of these might be better than either alone. GIRARD and GIRARD (p. 971) found M & B 693 to be as efficacious as plague serum in the treatment of infected mice and guineapigs.

BHARGAVA (p. 971) gives advice on the protection of coolie workers in an anti-plague campaign. JAMES (p. 972) considers that the vigorous measures taken prevented an outbreak in the Mawchi mines in India.

BABENUISHEV *et al.* (p. 972) show that fumigation of the burrows of ground squirrels with chloropicrin or calcium cyanide, though not entirely eradicating the fleas in the burrows, reduces their numbers. TIFLOV and POTAPOV (p. 973) found that the majority of all species of rodent fleas in Russia migrate from burrows of field rodents when these are abandoned. TROTTER (p. 973) remarks on the successful use of poison bait and other anti-rat measures in Hawaii.

The Eastern Bureau of the Health Organisation of the League of Nations (p. 973) reports that in India plague has steadily declined in the last 10 years. The same is true of the Dutch East Indies, where the decline has coincided with mass inoculation by Otten's living avirulent vaccine. To be successful, fumigation should be repeated every 3 months. ROSIER (p. 974) attributes the decline of plague in Java, of which he gives figures, firstly to the use of the living vaccine of Otten, of which over 6 million inoculations have been given since 1935, and secondly to the campaign of house improvement. SCHÜTZE (p. 974) reports on field trials of vaccines in Java. With heat-killed vaccine no superiority of virulent strains over avirulent, or of smooth over rough was seen, but for maximal protection of rats it is essential that the vaccine be grown at 37°C., at which temperature envelope antigen production is highest. In mice, however, envelope antigen is not so important. The superiority of smooth avirulent living vaccine depends on the capacity for survival in the inoculated animal, a rough strain is more quickly suppressed.

Vogel and Riou (p. 975) show that in Madagascar the incidence of plague has markedly decreased in 1936–1937 compared with previous years. During 1937 vaccination with the living EV vaccine was performed on 77·5 per cent. of the population of 1,051,395 and the authors attribute the decline of the disease to the use of this vaccine. Anchezar (p. 976) gives the characteristics of the EV strain of *P. pestis*.

Sokhey (p. 976) gives the desiderata for the preparation of vaccines of killed plague bacilli and quotes an experiment in which treatment with antiplague serum gave promising results. The composition of the rat population of Bombay is changing: *R. rattus* and *R. norvegicus* are now highly resistant to plague, but *Gunomys varius* is increasing and is highly susceptible. He gives the growth requirements of *Past. pestis*. He also (p. 977) states the minimum mouse-protection doses of vaccines prepared by different methods and at different temperatures.

In Brazil, apart from deratization on the usual lines, vaccination with heat-killed vaccines has been used. Barreto (p. 977) reports that the disease has been relatively mild. In plague epidemics Silvetti (p. 977) shows that vaccination produces an undesirable negative phase and prefers to rely on provision for the sick, segregation, disinfection and deratization. Bistrenin (p. 978), for the preparation of sugar vaccine, autoclaves the sugar at *p*H 6·8, afterwards neutralizing with sterile alkaline solution. In this way the production of acid is prevented.

Bahr (p. 978) shows that the ratin bacillus has undergone little change in 31 years. Rats which survive ratin ingestion may be killed by means of the squill preparation ratinin, though this is not satisfactory for field mice. *C. W.*

Yang (Y. N.), Landauer (E.), Koo (C. K.) & Lin (P. C.). **Plague Work in Fukien, China, December 1935 to November, 1936.**—*Chinese Med. Jl.* 1939. Jan., Feb., Mar., Apr. & May. Vol. 55. Nos. 1, 2, 3, 4 & 5. pp. 55–73; 162–173; 262–275; 383–390; 479–487. With 24 figs.

In the pandemic of plague which started from Hong Kong in 1894, the neighbouring province of Fukien, situated opposite the island of Formosa was soon involved. Its two principal ports, Amoy and Foochow, became infected in 1894 and 1901 respectively. The present account relates to plague work during 12 months, which was started as a result of a severe outbreak of plague at the end of 1935 in Southern Fukien. It deals in a series of chapters with the organization of the provincial plague prevention bureau, an epidemiological survey, sanitary surveys of endemic plague areas, preventive measures and finally with a rat and flea survey of Lungyen, a town 100 miles up river and the headquarters of the group of workers.

In early days the plague infection was dispersed by rats and fleas in merchandise from the seaports by river into the interior. As most of the province is mountainous, the bulk of inland trade is confined to the waterways and the seaports provided primary endemic plague foci. Some interesting information has been collected of the history of plague development in certain limited areas. On entering such an area the result of infection was "a few sporadic cases or a mild outbreak." "More or less severe outbreaks followed in subsequent seasons with or without an interval of from one to three or four years.

After a decade of periodical recurrences of epidemics the outbreaks became milder again and in some places the disease disappeared altogether." But the interepidemic intervals continued to show sporadic cases and these were very important as an index of the prospects of epidemic outbreak. The rat communities on farms are rapidly exterminated in an epizootic and as these farms are separated from one another by almost impossible country, the epizootic may die out completely. But, on the other hand, transmission to another area might be effected, human sporadic cases would occur and the stage was then set for appearance of a small epidemic. Even so-called sporadic cases, however, must really be regarded as true miniature epidemics. Villages likewise may be the source of reinfection of a new and susceptible rat population in the towns. No differences could be observed in the way in which plague attacked human beings in the matter of sex, age or occupation. It does not seem to have been possible in the present investigation to obtain actual data of length of the incubation period of plague but from the fact that " dead rats were always found 4–6 days prior to the onset of the disease in human cases, the incubation period must be within this limit." Pneumonic plague is rightly feared in China but in the present year's epidemic only two cases of secondary pneumonia occurred in two bubonic cases. One of these gave rise to a small pneumonic epidemic of 4 persons in the same house.

A most interesting, albeit melancholy, account is given of present-day sanitation in many Chinese towns. Water supply is from wells, often in a state of disrepair ; sewers are open channels ; latrines are of varied types, and human faecal matter is much in request for fertilizer; garbage disposal is still frequently primitive and streets and housing are not sanitarily satisfactory. It may be said indeed that conditions generally are highly favourable to rat infestation, although this must be ascribed very largely to the impoverishment of the people. Certain differences are noted in the architecture of North and South Fukien, but over the whole province rat infestation is almost a regular state of affairs.

Preventive measures adopted for the control of plague follow the usual lines. Rat burrows were treated by the calcium cyanide method of fumigation and a house-to-house distribution of poison bait was made by the fumigators. Rat-proofing measures for shops took into account the protection of (1) bulk stock, (2) replenishment stock, and (3) retail goods. " Economically this division of rat-proofing methods into three classes . . . . was a conspicuous success that kept the total costs of the campaign very low." The methods also constituted a welcome insurance for the shopkeeper of protection of his goods " against spoilage and consumption by rats." In Lungyen great help was accorded to the work of rat-harbourage elimination and rat proofing by military as well as civil authorities. "The staff of the 10th Army assisted in the conducting of several clean-up campaigns each taking nearly a week's work of over one hundred inspectors." Rules governing the construction of new houses were discussed with the local authorities as regards method of enforcement and distributed in pamphlet form to the local building constructors.

A chapter on the rat and flea survey of Lungyen is the last of this series. A total of 2,345 rats, trapped during the year, was submitted to examination and the species of fleas found were :—*X. cheopis*, *L. musculi*, *M. anisus* and *N. nicanus*.

The account of plague work given in these articles shows evidence of excellent organization and very praiseworthy co-operation between all the authorities concerned. *W. F. Harvey.*

Rao (S. Raghavender). **Some Observations on Plague in Calcutta.**—*Calcutta Med. Jl.* 1939. Aug. Vol. 36. No. 2. pp. 89–105. With 5 figs.

The long-continued absence of epidemic plague from Calcutta is well known but has scarcely been fully studied. Sporadic cases of plague have occurred at infrequent intervals and attract but little attention. It was the occurrence of such a case in a bustee or group of huts which led the author to take up a more intensive investigation of the problem especially as regards the possibility of importation. His work has shown that even in such a very small population as that of a bustee, deaths may be occurring which may be plague and that the known factors of rat density, flea infestation, climatic conditions of temperature and humidity in Calcutta are no less unfavourable to the development of plague than elsewhere. He found no evidence to support the contention that the case of plague in question was imported and he is led to suggest that plague cases would be found if looked for and that some systematic examination of the rodent population, including the bandicoot as well as the smaller rodents, is desirable. Such an examination would naturally take up the question of the relative immunity of the Calcutta rodent population to plague infection.

W. F. H.

Boletin Sanitario. Buenos Aires. 1938. Nov. Vol. 2. No. 11. pp. 802–815. With 5 figs.—Antecedentes epidemiologicos de la peste en los puertos de la Republica Argentina. [**Epidemiological History of the Entry of Plague in the Ports of the Argentine.**]—Informe presentado a la X Conferencia Sanitaria Panamericana por el Presidente del D.N. de H., Dr. Miguel Sussini.

It was in 1899 that plague was introduced to South America by a Dutch steamer calling at Montevideo with a cargo of rice from India. From Montevideo the cargo, after transference to another steamer, was taken to Asuncion in Paraguay and by this time an epizootic of plague had broken out and there were also plague cases. The further progress of plague by river brought it to Rosario and then to Buenos Aires in the Argentine Republic. Spread to the ports of the Argentine generally was then only a matter of time. In these ports the difficulties of conducting an anti-plague campaign of sanitation and deratization are very great. They proceeded on the well-known lines and have led to an extraordinary diminution of rats in the ports which has in some cases reached 70 per cent. *W. F. H.*

Boletin Sanitario. Buenos Aires. 1938. Nov. Vol. 2. No. 11. pp. 816–822. With 1 fig.—Peste rural en la Republica Argentina. [**Sylvatic Plague in the Argentine.**] Informe presentado a la X Conferencia Sanitaria Panamericana por el Presidente del D.N. de H., Dr. Miguel Sussini. (Preparado por los Dres. C. A. Alfarado y J. M. De la Barrera.)

In the Argentine as in other countries the type of plague known as sylvatic has assumed importance because of its tendency to appear in

epizootic form and to spread over a wide extent of country. The epizootic makes its appearance during the winter and fades out in spring. It reappears again the following winter. The infection in animals is of subacute type and death may take place without much in the way of macroscopic lesion. Among the animals affected are : *Microcavia australis*, *Galea musteloides*, *Graomys griseoflavus* and, to a less extent, *Lepus europaeus*, *Hesperomys murillus* and *Sylvilagus brasiliensis*. The parasitic *Siphonaptera* are represented by species of the genera Parapsyllus, Thopallopsyllus and Hectopsylla.

One of the features of sylvatic plague is the small amount of human infection which is experienced. This, however, is somewhat offset by the tendency to production of pneumonic plague. *W. F. H.*

Castellani (Aldo). **Brief Note on a Culture Medium used in the Differentiation between** *B. pestis* (*Pasteurella pestis*) **and** *B. pseudo-tuberculosis rodentium* (*Pasteurella pseudo-tuberculosis rodentium*).—*Jl. Trop. Med. & Hyg.* 1939. June 1. Vol. 42. No. 11. p. 158. With 1 coloured plate. [Summary appears also in *Bulletin of Hygiene*.]

The author proposes a method for cultural differentiation of *P. pestis* from *P. pseudotuberculosis rodentium* by their action in media containing glycerol and rhamnose. The latter produces acidity in one or both of these whereas the former does not. The composition of the medium used is : Rhamnose 1 gm., glycerol 4 cc., litmus solution (Kübel and Tiemann) 5 cc., agar 2 gm., water to 100 cc. *p*H 7·2.

In this *P. pestis* effects no change in colour, unless, perhaps, to darken the blue, whereas ten out of eleven strains of *P. pseudotuberculosis rodentium* changed the colour to red in two days, and the remaining strain gave a similar, though delayed, reaction. The author states that all strains of the latter do not ferment both ; some produce acidity in glycerol but not in rhamnose, some in rhamnose and not in glycerol.

H. H. S.

Rao (M. Sadashiva). **The Nutritional Requirements of the Plague Bacillus.**—*Indian Jl. Med. Res.* 1939. July. Vol. 27. No. 1. pp. 75–89. [23 refs.]

Very interesting work has been done in late years on the nutritional requirements of microorganisms. The autotrophic bacteria, regarded as on a low level of evolution, can assimilate carbon dioxide, whereas the heterotrophic bacteria are dependent on ready elaborated carbon compounds. The most primitive types of bacteria again can assimilate their nitrogen from inorganic sources ; at a higher level amino-acids, such as tryptophane, may be necessary for nutrition and at the highest level of development not only are a series of amino-acids necessary but also certain accessory growth factors or " bacterial vitamins " are indispensable. Pathogenic bacteria belong mostly to the last class. The present study is directed to determine : whether the plague bacillus can be grown in a simple chemically defined medium ; what accessory factors are necessary ; any nutritional differences between virulent and avirulent strains ; what amino-acids are essential as growth factors. The principal findings are :—(1) Three amino-acids,

proline, phenylalanine and cystine are indispensable while the presence of glycine though not essential, is stimulatory. (2) Accessory growth factors have no essential rôle in the nutrition of the plague bacillus. (3) Virulent and avirulent strains show little difference in nutritional requirements. *W. F. H.*

Tumansky (V.). Influence de *B. proteus vulgaris* sur le *B. pestis*. [**Influence of *Proteus vulgaris* on *Past. pestis***.]—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1938. Vol. 17. No. 1–2. [In Russian pp. 20–27. French summary pp. 27–28.]

Proteus vulgaris is antagonistic to *P. pestis* which finally disappears if the two organisms are cultivated together. This action of the Proteus organism is exercised only in living culture. Neither dead cultures nor bouillon culture filtrates possess any such effect. The virulence of the plague bacillus is at the same time diminished. It is understandable how plague bacilli disappear in decomposing human or animal bodies, for the putrefaction is to a considerable extent due to Proteus organisms. In a body kept at 28°–30°C. the virulence of plague diminishes very rapidly, much less rapidly at 12°–15°C. *W. F. H.*

Russo (Canio). Persistenza di vitalità e virulenza del bacillo della peste nel midollo osseo. [**Viability and Virulence of Plague in Bone Marrow.**]—*Rendiconti Istituto di Sanità Pubblica.* Rome. 1939. Vol. 2. Pt. 1. pp. 197–200.

The importance of the examination of the bone marrow in plague rats, especially if long dead, has been abundantly confirmed. In these experiments the author has been able to show how long the plague bacillus may maintain itself alive and virulent in rat or guineapig cadavers. He found experimentally that this time extended to 8 or 10 months and that even after 10 months pure cultures of the plague bacillus, without contamination by the putrefactive Proteus organism, could be obtained from the bone marrow of the femur. The vitality and virulence of the plague bacillus, moreover, had not undergone any modification during this time. Such survival of the plague bacillus permits acari, which live on the bodies of mummified rats, to become infected. *W. F. H.*

Breininger (D.). Action de la chlorure de chaux sur le bacille pesteux. [**Action of Calcium Chloride on the Plague Bacillus.**]—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1938. Vol. 17. No. 1–2. [In Russian pp. 116–119. French summary p. 120.]

A 2½ per cent. solution of calcium chloride kills a bouillon culture of plague in 10 minutes. Scraps of cloth infected with a 24-hour culture exposed to the action of a 10 per cent. solution are sterilized in 10 minutes. A 20 per cent. solution does not kill plague bacilli in sputum after 24 hours' action nor in the dead bodies of spermophils after 36 hours. *W. F. H.*

Shmelev (K.) & Fedorov (V.). **Disinfectants and the Plague Bacillus.**—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1938. Vol. 17. No. 1–2. [In Russian pp. 104–111. English summary p. 111.]

Favorissova (B.). Modifications morphologiques du *B. pesteux* sous l'action du bactériophage. [**Morphological Modifications of the Plague Bacillus by Bacteriophage.**]—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1938. Vol. 17. No. 1–2. [In Russian pp. 11–17. French summary pp. 17–19. With 35 figs.]

The action of bacteriophage in inactivated human serum and bouillon cultures on virulent *P. pestis* was studied in films prepared every five minutes during the first 12 hours of incubation and then at intervals of 24, 48, 72, 96 and 121 hours. The atypical forms which made their appearance during 121 hours in the serum culture were describable as rod-shaped, round, pyriform, semilunar and curved. In bouillon the same transformations took place but so rapidly that at the end of two hours there was nothing to be seen at all in the films.

W. F. H.

Russo (Canio). La propagazione del bacillo della peste in rapporto alla metamorfosi di insetti coleotteri ed acari. [**Propagation of the Plague Bacillus in Relation to the Metamorphoses of Coleopterous Insects and Acari.**]—*Rendiconti Istituto di Sanità Pubblica.* Rome. 1939. Vol. 2. Pt. 1. pp. 175–196. [25 refs.]

Flesh eating flies of the genus Sarcophaga offer favourable conditions to the multiplication and diffusion of the plague bacillus at all stages of their metamorphosis. The author has found that quite a number of invertebrates, that is to say, the ectoparasitic and endoparasitic insects of the muridae and some arthropods can contract plague infection and become true reservoirs of plague bacilli and thus the intermediate sources for the conveyance of infection. *W. F. H.*

Roberts (J. I.). **Rat and Flea Conditions in a Rural Endemic Plague Area in Kenya.**—*Jl. Hygiene.* 1939. July. Vol. 39. No. 4. pp. 355–360. With 4 figs.

Tables give the results in detail of a " survey of rats and fleas in the main endemic plague area of Kenya . . . . over a further period of two years " and graphs deal with rainfall and human plague, numbers of *R. rattus*, and *X. brasiliensis* index, rat plague and human plague for the periods 1934–5, 1935–6 and 1936–7. These figures and graphs demonstrate that " the factor exercising the greatest influence on the incidence of plague is the *Rattus* population density " ; that the period of maximum incidence of fleas per rat " agrees closely with the higher incidence of plague " ; that " endemic areas have a much higher *Rattus* density and higher breeding rates than plague-free areas " and that " the incidence of plague both in man and rats is more closely associated with rat population than any other factor." *W. F. H.*

Roberts (J. I.). **The Relationship of Field Rodents to Plague in Kenya.**—*Jl. Hygiene.* 1939. May. Vol. 39. No. 3. pp. 334–344.

The importance of an increase in the breeding rate and in the mortality rate of field rodents has been greatly stressed of late years in

connexion with the spread of plague epizootics to distant places. This sylvatic plague question is a serious one for public health authorities. In Kenya the matter has been approached essentially from the point of view of proof firstly of the fact and secondly of its importance for human plague. A high mortality among field rodents in the Rift Valley, coexistent with an outbreak of disease among sheep, was shown not to be plague but what is now known as Rift Valley fever. Bacteriological examination for plague in wild rodents over many years has not shown a single " field rat naturally infected with the disease." During 1930–31 when large numbers of field rats were dying in and around Nairobi, a total of 2,750 field rats—*M. coucha*, Arvicanthus and Otomys—were examined with negative results. In transmission experiments during an outbreak of plague, whereas *X. cheopis* and *X. brasiliensis* from rats in huts were easily shown to be vectors, experiments with 469 *Ctenophthalmus cabirus* and 816 *Dinopsyllus lypusus* acquired from field rats, " in the same area also suffering from an epizootic disease," proved negative.

In the summary to this article one of the conclusions reached for Kenya is that : " In the absence of any confirmatory bacteriological evidence to prove that epizootics among field rats are caused by *B. pestis* and the failure to find natural infection among field fleas together with the knowledge of their aversion to feed on man and lack of opportinity to develop such tastes, the theories that field rodents and their fleas " are definitely concerned in the spread of plague or in the initiation of outbreaks of plague " appear to be fallacious." *W. F. H.*

VAN CAMPENHOUT (J.). La peste au Congo Belge en 1937. [**Plague in the Belgian Congo in 1937.**]—*Bull. Office Internat. d'Hyg. Publique.* 1938. Dec. Vol. 30. No. 12. pp. 2747–2749. With 1 map.

The only focus of plague (sylvatic) of the Colony, at Lake Albert, produced 6 cases in 1937 of which 3 were bubonic, 2 pneumonic and 1 septicaemic. It was found that the rat *Mastomys ugandae* is a carrier, but *E. rattus rattus* plays no part in the endemic. Of the fleas found on rodents *Xenopsylla cheopis* and *brasiliensis* are in a large majority, while Leptopsylla, Dinopsyllus, Ctenocephalus, Sarcopsylla, Ctenopthalmus and Neopsylla have also been met with. *W. F. H.*

LOBANOV (V.) & FEDOROV (V.). **On Pathogenesis of Experimental Plague in Southern Gerbils** (*Pallasiomys meridianus* **Pall.**)—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1938. Vol. 17. No. 1–2. [In Russian pp. 57–70. English summary pp. 70–71.]

The authors met with a distinct seasonal difference of reaction in gerbils which were experimentally infected with plague. From April to July the type of disease induced was comparatively benign and remained localized. The micro-abscesses produced became encapsuled, absorbed and finally replaced by connective tissue, cicatrizing elements. From July to October on the other hand the plague induced was to a large extent acutely septicaemic and generalized.

W. F. H.

Korobkova (E.), Favorissova (B.) & Krainova (A.). Sur la valeur diagnostique des réactions sérologiques dans l'immunité antipesteuse chez les spermophiles. [**Diagnostic Value of Serum Reactions in Spermophils.**]—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1938. Vol. 17. No. 1–2. [In Russian pp. 72–86. French summary pp. 86–87.]

If the serum of spermophils gave retrospectively an agglutination reaction to plague, this would be of assistance in marking down a suspected enzootic area, especially if this reaction occurred in the older animals and not in the younger. Experiments in the laboratory on 200 spermophils caught in a non-plague area showed :—(1) The serum of healthy spermophils often agglutinated formolized cultures of the plague bacillus in dilutions between 1–10 and 1–40. (2) Immunization of spermophils with 3 injections of weakly virulent living plague on 3 successive days resulted in production of a feebly agglutinating serum (1–10 to 1–50) by the second week and disappearance of the agglutination after 25 days. (3) Spermophils immunized with living bacilli are protected against a very severe test dosage. (4) Specific agglutinins appear in the serum only after numerous injections of living bacilli, but a single injection is sufficient to confer immunity.

The conclusion drawn is that agglutination reactions are not likely to prove of much value in determining the epidemiology of a district.

W. F. H.

Gibbons (R. J.). **Survey of Rocky Mountain Spotted Fever and Sylvatic Plague in Western Canada during 1938.**—*Canadian Public Health Jl.* 1939. Apr. Vol. 30. No. 4. pp. 184–187.

A co-operative survey has been undertaken for the determination of the existence and the danger of occurrence of the two diseases Rocky Mountain spotted fever and wild rodent or sylvatic plague in the Canadian provinces of Alberta and British Columbia. No plague infection was found " in the 3,569 wild rodents and 7,582 rodent fleas examined." That is matter of congratulation but does not imply that there should be any relaxation of vigilance, as " the history of the progress of the infection northward and eastward through the western section of the United States " makes probable its extension to Western Canada within the next few years.

Investigation of the reports of Rocky Mountain spotted fever revealed that though the first case had only been notified from Alberta in 1935 and from British Columbia in 1936, " fully authenticated cases had occurred as early as 1923 in Alberta and 1917 in British Columbia." Laboratory examination of specimens of the ticks *Dermacentor andersoni* and *D. variabilis* for *Rickettsia dermacentroxenus* were negative, although the ticks had been collected in large numbers " in the immediate vicinity where cases had occurred." In the course of these examinations " several specimens were found to be infected with virulent strains of *P. tularense* " [*sic*]. *W. F. H.*

Roudabush (Robert L.). **Survival of the Tropical Rat Flea in United States.**—*Science.* 1939. Jan. 27. Vol. 89. No. 2300. pp. 79–80.

Jellison (William L.). **Sylvatic Plague : Studies of Predatory and Scavenger Birds in Relation to its Epidemiology.**—*Public Health Rep.* 1939. May 12. Vol. 54. No. 19. pp. 792–798.

It seems quite possible that flesh-eating birds, especially those depending on rodents " as an important source of food supply," might be an important factor in the spread of plague. Again, birds might be agents of transportation to a distance of plague infected fleas. The booby owl for example " is a constant companion of the ground squirrel occupying the same burrows with him." The field and laboratory observations reported here were made in a plague epizootic area. Predatory species of birds were found to transport flea-infested rodents and to serve as accidental hosts of rodent fleas. Experimentally " casts from predatory birds fed plague-infected guineapig tissue were consistently infectious." In general no very positive conclusion seems to have emerged from these studies.

W. F. H.

Eskey (C. R.) & Haas (V. H.). **Plague in the Western Part of the United States. Infection in Rodents, Experimental Transmission by Fleas, and Inoculation Tests for Infection.**—*Public Health Rep.* 1939. Aug. 11. Vol. 54. No. 32. pp. 1467–1481.

Ground squirrel (*Citellus beecheyi*) infection was discovered within 10 years of the occurrence of plague (1900) at San Francisco in nine Californian counties. In 1934 epizootics occurred in the Sierra Nevada mountainous area, since which time systematic investigation has resulted " in the discovery of foci of wild rodent plague in 9 of the far western States exclusive of California." So far it has not been found in the great plains area east of the Rocky Mountains. The absence of domestic rats in most of this affected territory accounts for the absence of epidemics. More than " 50 different species of fleas have been found on the western rodents," ground squirrels with an average of 20 per animal and marmots with over 10 fleas each. Nor does the number found on the animal represent the total, for fleas also occupy the nests of rodents. It is not probable that this applies for very long, however, to the abandoned nest. In testing experimentally with fleas for the degree of infectiousness of plague infected guineapigs, this was found to bear a close relationship to the degree of bacteriaemia present. Thus no flea was found to be infected by blood which did not give a culture of *P. pestis*. There did not seem to be much difference between wild rodent fleas and domestic rat fleas in the readiness with which they became infected. Once the flea was proved infected by transmission its average length of life was only 3·2 days, a figure which held good for fleas of different species, but a " certain period must elapse for the extrinsic incubation before the bites were infectious." That period varied from 5 to as long as 130 days, thus demonstrating that fleas may harbour virulent plague in the gastrointestinal tract for a long time, a fact which is of the highest importance " in determining their efficiency as vectors."

The determination of the existence of plague infection in the wild rodents of a territory has now become a regular epidemiological procedure. Inoculation of guineapigs with the bodies of captured fleas has been found to be a much more effective means of finding rodent plague infection than by systematic examination of the killed animals themselves. An important discovery was that the parasites

killed with hydrocyanic gas were much more infective than if killed with chloroform or ether, due to attenuation of the plague organism brought about by the latter. Thus cyanide gas became substituted for chloroform in killing the parasites before removing them from their hosts. A 2 per cent. salt solution is used in which to transmit the fleas to the testing laboratory: it inhibits the growth of secondary bacteria, prevents putrefaction and has no deleterious action on *P. pestis*. "If a specimen contains a great many fleas they are usually divided into lots of 50 for inoculation." *W. F. H.*

Devignat (R.). L'utilisation du milieu de Broquet pour la recherche de la peste des puces. [**Broquet's Medium for Examination of Plague Fleas.**]—*Ann. Soc. Belge de Méd. Trop.* 1938. June 30. Vol. 18. No. 2. pp. 215–219.

Originally, suspected fleas were placed in 60° alcohol which is excellent but costly; then 10 per cent. phenol and finally pure phenol, which had the advantage of clearing the fleas for examination, were used. All these media, however, destroyed organisms so that, although usable for identification of flea species, no bacteriological examination of the fleas could be made. Broquet's medium, with the composition calcium carbonate 2, glycerin 20 and distilled water 80, acts as clearing agent for fleas, ensures a survival of plague bacilli for several days and prevents putrefaction. In the village which is undergoing its monthly deratization, the fleas recovered from the killed rats are placed in Broquet's medium; a medical orderly cuts off the legs of the rats with a pair of scissors, while another obtains femoral bone marrow with a syringe and emulsifies it in a small flask containing 5 cc. of sterile normal salt solution. Lastly a guineapig is inoculated subcutaneously with 1 cc. of the emulsion. If now on arrival at the laboratory of the district the guineapig dies of plague, it remains possible to isolate a strain of plague from the fleas which have been collected from the same place, on the same day and from the same rats.

W. F. H.

Devignat (R.). L'utilisation du milieu de Broquet pour la recherche de la peste des puces. (Note complémentaire.) [**Broquet's Medium for Examination of Plague Fleas.**]—*Ann. Soc. Belge de Méd. Trop.* 1938. Dec. 31. Vol. 18. No. 4. pp. 543–545.

This note is supplementary to the original communication (above) and shows that plague fleas can be preserved in the medium up to six days. *W. F. H.*

Murdock (John R.). Peste pulmonaire à Riobamba (Equateur). [**Pneumonic Plague at Riobamba (Ecuador).**]—*Bull. Office Internat. d'Hyg. Publique.* 1939. June. Vol. 31. No. 6. pp. 1022–1023.

This account is of a sharp short epidemic of pneumonic plague lasting only a few weeks and giving rise to 15 deaths out of 16 cases. After the first case had been admitted to hospital there followed rapidly the contact infection of nursing sisters, attendants and doctor. All were cases of pneumonia and only one nursing sister survived. A diagnosis was made by exhumation of one of the fatal cases, removal

of a portion of rib and examination of bone marrow by film and injection of a guineapig. Direct contacts and indirect contacts were isolated separately. No new cases had occurred at the time of reporting for 3 days. *W. F. H.*

FARINAUD (M.). Un cas d'association du bacilli de Yersin à un streptocoque. [**Association of the Plague Bacillus with a Streptococcus.**]—*Bull. Soc. Path. Exot.* 1939. Apr. 4. Vol. 32. No. 4. pp. 443–445.

The association in the described case was of a streptococcus, present alone in buboes in the early stage of the disease, with the plague bacillus in the later. This is the reverse of an association which is sometimes found in pneumonic plague, where a pneumococcus may appear on the scene along with the plague bacillus, but only at a late stage of the disease as a secondary infection. A child of 25 months had been ill for 5 days and had suffered from fever and diarrhoea for 3 days before admission to hospital. A bubo as large as a hen's egg was present in the axilla. Two other buboes made their appearance, one in the neck and one in the groin. The temperature reached 40·6°C. and the child died in hyperpyrexia. Several gland punctures were performed, one of them about 40 minutes before death. The material of these punctures showed streptococcal chains in abundance and a gradual appearance of gram-negative bacilli although without sign of bipolar staining. Inoculation of a mouse and a guineapig with the gland juice withdrawn just before death caused the death of the animals with acute plague, while that which was taken after death when also injected into a mouse and a guineapig, produced no symptoms at all. The streptococcus isolated from the puncture material was of low virulence, for it only killed mice after 8 days. This case is claimed as one of association of a streptococcus with the plague bacillus.

W. F. H.

i. DURAND (Paul). Action du Dagénan dans l'infection pesteuse. [**Dagenan in the Treatment of Plague.**]—*Arch. Inst. Pasteur de Tunis.* 1939. Mar. Vol. 28. No. 1. pp. 96–106.

ii. ——. Action du 693 M.B. dans l'infection pesteuse.—*Bull. Soc. Path. Exot.* 1939. Mar. 8. Vol. 32. No. 3. pp. 267–271.

i. These experiments were carried out on mice with para-aminophenyl-sulphamido-pyridine, shortly known as dagenan or M & B 693. It was incorporated in a paste made of crushed barley, flour and sweetened condensed milk which, after drying, was suspended in pellet form in a glass jar containing 3 or 4 mice. An untreated mouse, if inoculated subcutaneously with plague, dies in a few days, shows gelatinous oedema at the site of inoculation and sometimes a regional bubo. The blood and the organs swarm with bacilli which are evident microscopically. Cultures are easily obtained from the blood or organs and inoculation of spleen or blood into a fresh mouse kills it in a few days.

Summarized, the results were :—Dagenan, of which about 2 mgm. per gm. of animal was ingested daily, was fairly well tolerated. The mice inoculated on the 5th or 6th day of treatment, which was continued for 6 to 17 days later, survived a test of 10,000 lethal doses. Almost all the mice tested with 20,000 to 300,000 lethal doses succumbed

but were, none the less, sterilized, for no plague bacilli could be found microscopically, by culture or by inoculation in blood organs. If, however, the daily dosage were reduced to one-quarter of that mentioned protective action and sterilization were only imperfect. It was interesting to find also that most of the mice saved by dagenan from plague infection had acquired considerable immunity to infection and that this immunity was greater the larger the first infecting test injection.

ii. This appears to be a rather shorter version of the above paper.

W. F. H.

Schütze (Harry). **Chemotherapy in Plague Infection.**—*Lancet.* 1939. Feb. 4. pp. 266–268.

The drugs which have been tested experimentally in white rats and mice for their efficacy against plague infection are:—(1) 2-sulphanilylaminopyridine (M & B 693), (2) soluseptasine, and (3) a diaminodiphenylsulphone glucoside ("sulphone"). White rats are rather resistant to plague and very large doses of plague bacilli are required to obtain 100 per cent. mortality. With a dose of 100,000 to 1,000,000 organisms subcutaneously as many as 40 per cent. of the rats may survive. White mice, on the other hand, will all succumb to a dose of about 100 organisms. The two types of animal are useful in experimenting with the synthetic drugs, to bring out essential differences between them. All three drugs were given before or with the test dose, subcutaneously or by the mouth, and all three produced definite therapeutic effects: "sulphone in mice and soluseptasine in rats having a slighter protective value and M & B 693 being more potent than either in both rats and mice." At the same time an experiment was carried out with anti-plague serum injected simultaneously with the test dose, but in the opposite flank, that is to say essentially as a prophylactic. The serum had been prepared by injection of horses with the living virulent Otten strain of plague bacilli. The larger doses of serum protected 5 out of 6 rats and 5 out of 6 mice, thus giving results comparable with those achieved in rats by M & B 693." A combination of serum with drug may possibly "prove superior to either alone." *W. F. H.*

Girard (G.) & Girard (M.). Remarquable efficacité du corps "693" M.B. *p. aminobenzène sulfamido-pyridine* dans le traitement de la peste expérimentale. [**Remarkable Efficacy of M & B 693 in Experimental Plague.**]—*Bull. Soc. Path. Exot.* 1939. May 10. Vol. 32. No. 5. pp. 480–482.

In these experiments, which are of a preliminary nature and will be published in detail later, M & B 693 was found to be as efficacious in both mice and guineapigs as plague serum. The mice received 10 cgm. in their food over 6 days, counting from the moment of infection. Experiments in the guineapigs were intentionally varied, but they received from 12 to 50 cgm. per day for 5 to 17 days without sign of poisoning. Control animals all died. *W. F. H.*

Bhargava (R. C.). **Protection of Anti-Plague Workers.**—*Indian Med. Gaz.* 1939. Feb. Vol. 74. No. 2. pp. 87–88.

The coolie worker in an anti-plague campaign should have some instruction in the mode of spread of plague and in the dangers

attendant upon his work. The dangers are, of course, from fleas and from the poison gas of disinfestation. It is recommended that his clothing should be white and so disposed that fleas can get no access to the skin anywhere, while he should wear a glove of thick rubber reaching from fingers to elbow as protection during cyanogassing. Coolies should work in batches of two so that one can keep a watch on the other for fleas. It is important likewise that hours of work should not be too long nor too continuous lest the workers become careless and off their guard against infected fleas.

A note to this article gives as simpler methods: " (*a*) protection of workers by inoculation with antiplague vaccine, (*b*) wearing of white overalls, and (*c*) smearing of the hands up to elbows and legs up to knee with kerosene-soap emulsion daily just before starting the work." If cyanogassing is done with calcid briquettes it carries little danger to either worker or public. *W. F. H.*

JAMES (E. R.). **The Incidence of Plague in Mawchi Mines and the Advantage of adopting Early Preventive and Prophylactic Measures.** —*Indian Med. Gaz.* 1939. June. Vol. 74. No. 6. pp. 347–348.

Plague was prevalent from December to the following February in the area for the first time. As the area was densely populated, rats fairly numerous and the camps overcrowded, it might have been expected that an epidemic, if it occurred, would be severe. The non-occurrence of such an epidemic may be reasonably ascribed to the vigorous anti-plague measures adopted. Only five cases of plague did occur. *W. F. H.*

BABENUISHEV (V. P.) & others. **Observations on the Fate of Fleas dwelling in the Nests of Ground Squirrels situated in the Regions that had been subjected to Entire Poisoning.**—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1937. Vol. 16. No. 3–4. pp. 467–474. [In Russian.] English summary. [Summarized in *Rev. Applied Entom.* Ser. B. 1939. Aug. Vol. 27. Pt. 8. pp. 170–171.]

" An account is given of investigations in 1935 and 1936 in 8 districts of the province of Ordzhonikidze (northern Caucasus) to determine whether fumigation of the burrows of ground squirrels [*Citellus pygmaeus*] with chloropicrin or calcium cyanide also destroys the fleas in them. The work was done in spring in localities in which all the burrows had been fumigated for two or three consecutive years; to ascertain the presence of fleas, the entrances of the burrows were opened and wads of cotton-wool were inserted into them and examined 24 hours later. Fleas occurred on 44 wads out of 4,474 inserted in the morning (0·9 per cent.) and on 20 wads out of 3,182 inserted in the evening (0·62 per cent.). It was shown, however, by examining nests from treated and untreated burrows that the numbers of fleas were reduced by fumigation. The average numbers per nest were 15·7 in fumigated burrows that still retained the straw plug and were covered with earth, 31·3 in fumigated burrows that had been opened for some time and may have been used by various small animals, and 49 in the untreated inhabited burrows.

" Fleas collected from the ground squirrels belonged to 14 different species, the abundance and local distribution of which are shown in a table. They included *Ceratophyllus* (*Oropsylla*) *ilovaiskii*, Wagn. and Ioff, which had not previously been recorded from the northern Caucasus."

Tiflov (V. E.) & Potapov (V. D.). **Migrations of Fleas of Ground Squirrels** *Citellus pygmaeus* **Pall.**—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1937. Vol. 16. No. 3–4. pp. 438–466. With 1 graph & 2 figs. [14 refs.] [In Russian.] English summary. [Summarized in *Rev. Applied Entom.* Ser. B. 1939. Aug. Vol. 27. Pt. 8. p. 170.]

" In view of the importance in the Russian Union of fleas of rodents in the dissemination of plague, and to supplement investigations by other workers, field observations on the migration of fleas from abandoned burrows of *Citellus pygmaeus* and on their survival under natural conditions were carried out in Western Kazakstan from 1st August to 1st December 1934, and from 4th March to 15th November 1935. For this purpose, all the ground squirrels over an area of 25 acres were caught, and the fleas that tried to leave 50 burrows were trapped. The traps consisted of a metal box containing water which the fleas entered through a tube ; the end of the tube was inserted into the entrance hole of the burrow. The results, which are tabulated, showed that all the species of fleas that occur on ground squirrels migrate from abandoned burrows. Species that have well developed eyes and infest the host itself (*Ceratophyllus tesquorum*, Wagn., and *Frontopsylla semura*, Wagn. and Ioff) markedly predominated, whereas those that have rudimentary eyes and inhabit the nests (*Neopsylla setosa*, Wagn., and *Ctenophthalmus pollex*, Wagn. and Ioff) were comparatively scarce. In 1934, most of the fleas migrated in August and only two in November. In 1935, the maximum number of fleas was caught in April, after which there was a sharp reduction, and only one flea was taken in July. After migration had ceased in July, 32 burrows were opened and examined for the presence of fleas ; 19 live individuals were found, in 7 burrows. A few rodent fleas were caught outside burrows in 1935, and observations on batches of *N. setosa* showed that many could survive on the soil for 24 hours and a few for 48 hours or more."

Hawaii, Territory of : Annual Report of the Board of Health for the Fiscal Year ended June 30, 1938 [Trotter (F. E.), Territorial Commissioner of Public Health]. [**Plague** pp. 182–205. With 9 graphs.]

Under the heading Plague this report includes a number of subsidiary reports of administrative type from the health officers, sanitary engineers and trappers of the plague service. A very interesting observation is made by the Territorial Commissioner of Public Health : " The principal advance made in plague control measures during the year was the discovery on the Island of Haui that the rat population could be reduced markedly by the proper application of a poison bait." The standard bait consisted of a piece of banana loaded with phosphorus. Other measures of limited application were the use of : (1) calcium cyanide dust ; (2) carbon bisulphide for exposed burrows, and (3) sodium arsenite solution for spraying. *W. F. H.*

League of Nations. Health Organisation. Eastern Bureau, Singapore. Annual Report for 1938. [**Disease Incidence. Plague in Countries and Ports.** pp. 13–27. With 10 graphs & 1 map.]

Under the head of Plague the subject of disease is treated for both countries and ports. In India the tendency of plague has been

towards a steady decline during the past 10 years. Mortality has fallen from 121,242 in 1928 to under 7,000 in 1938. It is a decidedly localized mortality, for more than half of it occurred in the United Provinces. The general curve for British India shows normal seasonal rise in January to a peak at the beginning of March and a minimum in June. Usually a secondary rise takes place in August. In the Netherlands Indies, as in British India, plague incidence shows a steady fall particularly in Java where a " marked decline in prevalence has coincided with the use of mass inoculation by OTTEN's vaccine." That vaccine is prepared from a non-virulent living strain of plague.

Fumigation as a raticide plague measure would seem to require repetition " at intervals of not more than three months if the number of rats and their harbourages is to be kept under control."

Under the heading " ports " it is now necessary to consider air- as well as sea-ports. " Owing to its importance as an air-port Allahabad was included in May 1937 in the list of ports from which telegraphic returns are being sent weekly." Besides Allahabad ports dealt with are Bombay, Cochin, Karachi, Rangoon, Bassein, Colombo and Pnom-Penh. " Pnom-Penh was the only port in the Far East to report the presence of plague during the year." *W. F. H.*

ROSIER (H. J.). Verslag betreffende de pestbestrijding op Java over het jaar 1937. [**Report on the Campaign against Plague in Java during 1937.**]—*Meded. Dienst d. Volksgezondheid in Nederl.-Indië.* 1938. Vol. 27. Supp. 2. pp. 85–166. With 11 graphs & 8 sketch maps & maps. English summary.

" In 1937 the general situation in regard to plague in Java remained satisfactory as the total incidence of 3,834 cases with 3,814 deaths compares favourably with the previous total of 6,227 cases with 6,187 deaths.

" The distribution over the different parts of the island was : West-Java 2,620 deaths, Mid-Java 1,150 and East-Java 44.

" The extent of the epidemic is practically unaltered. In some parts of the improved areas plague reappeared on a small scale, but this may be controlled without difficulty since deratting and restoration of improved houses goes on very easily.

" Primary pneumonic plague shows the usual percentage, 380 cases being spread fairly uniformly among the bubonic ones.

" Vaccination has been carried on with excellent results ; 1,804,234 inoculations have been effected without any accidents, most of these were re-inoculations. Since January 1935, 6,175,477 inoculations have been given in Java. Undoubtedly the steady decrease of the present epidemic since the peak-year 1934 (23,239 deaths) is to be attributed in the first place to the use of Otten's live plague-vaccine.

" Apart from this the policy of house-improvement is continued ; the progress of this year was 32,318 houses. Moreover, in the improved regions 56,260 houses have been built under anti-plague supervision."

SCHÜTZE (Harry). **Studies on** *B. pestis* **Antigens as Prophylactic Agents.**—*Brit. Jl. Experim. Path.* 1939. June. Vol. 20. No. 3. pp. 235–244.

Very interesting work on plague vaccines is at present being carried out at the Lister Institute London, Haffkine Institute Bombay and the Pasteur Institute Bandoeng in Java. Questions of importance which arise out of these experiments relate to the efficacy of living

and killed cultures ; agar and broth cultures ; virulent and avirulent strains ; rough and smooth varieties ; flagellar, envelope and somatic antigens ; incubation at 27° and 37°C. In Java notable trials have been made in the field with different vaccines on an alternate case basis, but one would like to see such trials conducted with strictly impersonal check and the most rigid insistence on the impossibility of unconscious selection. The present article is a noteworthy contribution to the subject and had better be abstracted, as far as possible in the author's own words, with anticipatory apology for any unintentional misreading :—

" Vaccines made from virulent, smooth avirulent and rough avirulent strains were prepared by growth in broth for 4 weeks and heating to 55°C. for 15 minutes, phenol to 0·5 per cent. being added. In the rat the test dose, a large toxic one, of 10 or more million bacteria, in mice a small invasive test dose of 100 or 200 bacteria was employed. There is a notable difference in the reaction of these 2 animals to subcutaneous infection with virulent plague. No superiority of virulent strains over avirulent or of smooth over rough could be demonstrated. For maximal protection of the rat it is essential that the vaccine be grown at 37°C., explained by the fact that at higher temperatures more envelope antigen develops in a plague culture and that plague prophylaxis depends, where the test dose is a large and toxic one, markedly on the presence of envelope antigen. In the mouse, where the test dose is a small and invasive one, envelope antigen appears to play a smaller part. It must be some other antigen which principally influences prophylaxis in the mouse and it must develop equally well in avirulent as in virulent, in rough as in smooth cultures, when these vaccines are killed. So far as the envelope antigen is concerned, and possibly other antigens, the superior prophylactic power of a smooth avirulent living vaccine depends on its capacity for prolonged survival in the inoculated animal. A rough strain is more quickly suppressed and has therefore less time to elaborate antigen. This would explain the well known fact that the smooth variant is superior to the rough in making living avirulent vaccine. In all four types, virulent or avirulent, rough or smooth, envelopes are equally well developed and vaccines made therefrom have similar E titres."

W. F. H.

Vogel (E.) & Riou (M.). Les maladies épidémiques, endémiques et sociales dans les colonies françaises pendant l'année 1937. Peste. [**Disease in French Colonies in 1937. Plague.**]—*Ann. de Méd. et de Pharm. Colon.* 1939. Apr. Vol. 37. Supplement. pp. 259–270. With 1 chart.

Plague in the French colonies seems to have run a more or less expected course in 1937. On the whole it has tended to regress. An account is given for each of the colonies of mortality statistics, sanitary preventive measures and plague vaccination. In Madagascar especially prophylactic vaccination has been on a large scale and this was carried out, as in 1936, on two occasions from 15th August to 30th November. The living EV vaccine was used and the total of vaccinations was 815,453 for a population of 1,051,395 inhabitants, or 77·5 per cent. A table of monthly graphs for the years 1933 to 1938 shows a marked decrease of cases of plague for 1936 and 1937 by comparison with the years 1933, 1934 and 1935. The results are claimed as showing the effect of the new method of vaccination on a large scale.

W. F. H.

Anchezar (B.). La infección experimental determinada por la Cepa EV. de Girard (=*Pasteurella pestis*, avirulenta). Su estudio bacteriológico y anatomo-patológico. [**Bacteriological and Pathological Study of the EV Plague Avirulent Strain.**]—*Folia Biol.* Buenos Aires. 1938. Sept.–Oct.–Nov.–Dec. Nos. 90–91–92–93. pp. 393–396.

Experiments conducted with the EV strain of Girard showed that (1) this strain, originally isolated from a case of bubonic plague, corresponded to *P. pestis* in all respects. (2) It was avirulent for laboratory animals. (3) The inoculation of large doses killed animals by toxaemia. (4) The strain had a marked selectiveness for lymph nodes and spleen; appeared for a short time in the blood and bone marrow and remained for longer time at the site of inoculation. [See also this *Bulletin*, 1939, Vol. 36, p. 313.] *W. F. H.*

Bombay: Report of the Haffkine Institute for the Year 1937 [Sokhey (S. S.), Director]. [**Plague** pp. 29–40; 47–48.]

Much of the research work noticed in this report has, naturally, already been published in journal form. The Director reviews the main points which have emerged from these researches. Plague vaccines made from broth cultures are more potent when incubated at 27°C. than at 37°C., but the reverse holds good for agar cultures. Those vaccines which are killed by the application of heat for 15 minutes at 55°C. are better than those where phenol or formalin are used. Special interest centres round the testing of the relative values of killed virulent plague vaccines and the same strain used in the avirulent but living state, in view of the extensive use of living plague vaccine in other parts of the world. It has been found, however, that the living avirulent strains tried have varied greatly in antigenic value from one of high protective value to one devoid of protective power. No measurable differences, on the other hand, have been found in the antigenic value of a large number of virulent strains. In a small trial of anti-plague serum, in which 124 cases were treated, 69 with serum and 55 controls with intravenous iodine, the controls exhibited a mortality of 65 per cent. compared with only 27 per cent. of the serum-treated.

Continuous observation has been going on for a very long time now in Bombay of the composition of the rodent population. It has been found that this population is changing. When plague was raging in 1907 " the rodents responsible for the spread of plague in the city were *Rattus rattus* and *Rattus norvegicus* which formed over 99 per cent. of the population." In 1937 these two species together form only about 70 per cent. and *Gunomys varius* makes up the remaining 30 per cent. *Gunomys varius*, moreover, is highly susceptible to plague infection, while the other two species are now highly resistant.

Some biochemical studies on the growth conditions of the plague bacillus have shown that its nutritional requirements are relatively simple and " it shows no dependence on accessory factors for growth." Three strains were used: a mixture of 8 amino-acids supplemented with phosphate buffer, sodium citrate and magnesium sulphate was experimented with to see which of the amino-acids was indispensable to growth. Proline and phenylalanine were wholly indispensable.

"In the absence of cystine only one strain grew. Glycine was nearly but not quite essential for two strains, while alanine appeared to be so for one strain." *W. F. H.*

Bombay: Report of the Haffkine Institute for the Year 1938 [Sokhey (S. S.), Director]. [**Plague** pp. 30–41; 51–52.]

The work carried out during the year on plague is very much on the lines of the previous report for 1937 (above). Perhaps the most interesting investigations are those on standardization and relative value of differently prepared vaccines. The method of statistical assessment of the results used is that "for estimating fifty per cent. end points employed by Reed and Muench (*Amer. Jl. Hyg.*, 1938, Vol. 27, p. 493)." Thus, for example, in minimum mouse-protective doses: (1) Broth vaccine incubated at 27°C. protected with 0·0025 cc., at 37°C. with 0·008 cc.; (2) agar vaccine incubated at 27°C. with 0·0851 cc. and at 37°C. with 0·0017 cc.; (3) live avirulent broth vaccine at 27°C. with 0·0025 cc. and at 37°C. with 0·008 cc.; (4) live avirulent agar vaccine at 27°C. with 0·0085 cc. and at 37°C. with 0·0017 cc. The effect of the mode of killing was also carefully estimated. *W. F. H.*

Barreto (João de Barros). A peste no Brasil nos ultimos 5 anos. [**Plague in Brazil in the Last Five Years.**]—*Arquivos de Hig.* Rio de Janeiro. 1938. Nov. Vol. 8. No. 2. pp. 347–374.

Plague has been endemic in Brazil, especially in the North East. The people of this region are chiefly occupied with the cultivation of corn, cotton and manioc. In the factories there are plentiful supplies of food for rats and the conditions are conducive to the propagation of fleas. During the period of the quinquennium 1934–1938 there were 1,393 cases of plague—208 in 1934, 822 in 1935, 314 in 1936, 35 in 1937 and 14 up to August, 1938. It was a relatively benign type of plague with mortalities of 35 per cent. in 1935, 39·5 per cent. in 1936, 43 per cent. in 1937 and 46 per cent. in 1938. This compares favourably with the ordinary mortality of 60 to 95 per cent. which attaches to bubonic plague. The rats involved were of domestic type—*norvegicus*, *rattus* and *musculus*—while the fleas were of the species *X. cheopis* and *brasiliensis*. An active campaign has been pursued against the disease and doctors and other workers have been put through a rapid special course before being detailed for the work. Methods of deratization and immunization have been of the usual description. The vaccines used were killed vaccines: (1) 2 cc. of a culture suspension from glycerin agar heated 1 hour at 65°C., and (2) a growth at room temperature of 48 hours on agar of *p*H 7·4, where the suspension was treated with formalin to give a final concentration of 1 : 2,000 and heated 1 hour at 50°C. For inoculation purposes a dilution was made to 1,000 million organisms per cc. and 2 inoculations were given at 7 days' interval. *W. F. H.*

Silvetti (Luis M.). Informe sobre una epidemia producida en Santa Ana (Tucumán) denunciada como peste septicemica. [**An Epidemic of Septicaemic Plague in Santa Ana (Tucuman), Argentine.**]—*Bol. Sanitario.* Buenos Aires. 1939. Mar. Vol. 3. No. 3. pp. 131–140.

The description given is of an epidemic which on bacteriological report was regarded as plague. The author attempted by exhumation

and examination of bone marrow to establish the diagnosis securely but failed owing to the advanced state of decomposition of the bodies. It is interesting to find that Silvetti condemns much of the routine procedure in plague epidemics as only of value for assuaging alarm. Prophylactic vaccine produces an undesirable negative phase condition and serum therapy allergic disease. In any case the protection afforded by these means is of very short duration. He would confine plague measures to providing for the sick, segregation of contacts, prophylactic disinfection and deratization. *W. F. H.*

Bistrenin (A.). **Modifications of Cane Sugar Water Solutions caused by Autoclave Sterilisation in Connection with the Preparation of AD Vaccine.**—*Rev. Microbiol., Epidémiol. et Parasit.* Saratov. 1938. Vol. 17. No. 1–2. [In Russian pp. 99–103. English summary p. 103.]

Autoclave sterilization of sugar solutions in media is apt to cause production of acid and so to alter the *p*H. This is especially the case with sugar in an alkaline solution. It is advisable therefore to sterilize the sugar at a neutral or slightly acid (*p*H 6·8) reaction and correct the *p*H afterwards by addition of a sterile alkaline solution. This procedure, of autoclave sterilization of a concentrated (60 per cent.) slightly acid sugar solution for the mass production of AD-sugar vaccine is a great saving of time. *W. F. H.*

Bahr (L.). Einige experimentelle Untersuchungen speziell an Wanderratten (*Epimys norvegicus*) mit " Ratin " und " Ratinin " (1907–1938). [**Experimental Investigations of the Effect of " Ratin " and " Ratinin " on Rats.**]—*Ztschr. f. Infektionskr. d. Haustiere.* 1938. Dec. 5. Vol. 54. No. 1/2. pp. 44–56. With 2 charts. [17 refs.]

A review of the results of the use of " ratin " from 1907 to 1937 is given ; 3,513 " ratin " culture strains, which had been freshly cultured from the blood or spleen of fatally infected rats, were used and were administered by mouth to 18,464 rats, giving a mortality of 60·8 per cent. A special set of 690 ratin stock cultures were more virulent and gave a mortality of about 90 per cent., with comparatively little variation from the average figure. It was shown by these experiments that the ratin bacillus had undergone little change in the whole 31 years of the trials. The stock cultures were carefully controlled as regards all their reactions, which were—for the carbohydrates—acid and gas in glucose, maltose, arabinose, rhamnose, dulcite, mannite and sorbite ; neither acid nor gas in lactose, saccharose, salicin, inosite and adonite. The sugar xylose gave variable reactions.

Rat extirpation was not confined to operations with " ratin " only but included the subsidiary squill preparation named " ratinin." In this way rats which had been able to survive " ratin " ingestion were finally killed. Ratinin was not satisfactory for the extermination of field mice, as they did not eat the preparations made.

Although several other countries conduct special campaigns against rats, Denmark is probably the first to introduce a law (1938) which makes such a campaign applicable at a given time to the entire country. The provisions of this law are set out in full in an appendix to the article. *W. F. H.*

TYPHUS GROUP OF FEVERS.

Précis of Abstracts in this Section.

General.—Sarkar (p. 981) records a rising titre for *Proteus OX2* in a European case of fever negative to *OX19* and *OXK* in India. Sixty-five of 438 sera tested at the King Institute, Guindy, were found positive to *Proteus OX19*, *OXK* or *OX2* in titres above 1/200. The majority were positive to *Proteus OX19*.

Blanc and Noury (p. 982) describe a temporary local skin immunity in rabbits inoculated with the virus of murine typhus and boutonneuse fever, which is not correlated with general and visceral immunity. Giroud, in two papers (pp. 982, 983), confirms this finding and also shows that local skin reactions are most marked in animals which are least susceptible to general infection.

Raynal and Fournier (p. 983) investigated the character of three strains of typhus virus isolated in Shanghai.

The same authors (p. 983) show that scrotal reactions may be caused by organisms other than Rickettsia. In Rickettsial diseases they are most common with the virus of murine typhus and occur only rarely with that of classical louse-borne typhus.

Nicolle and Simons (p. 984) show that the blood sedimentation rate is accelerated in experimental typhus.

Lestoquard (p. 984) uses iodine for the staining of Rickettsia and Giroud and Panthier (p. 984) describe the reaction of cells containing Rickettsia to Lugol's solution, which may be due to the staining of contained glycogen.

Cox (p. 984) has successfully cultivated Rickettsia in fowl embryonic membrane, and finds that on passage in this manner virulence is enhanced.

R. canis have been found in Beyrouth for the first time by Pigoury and Bernard (p. 985).

Herzig (p. 985) reports a Rickettsial disease occurring in volunteers on whom laboratory bred normal lice fed in Weigl's laboratory. Further work is necessary to decide the nature of the Rickettsia, but they resemble *R. weigli* and *R. quintana* of trench fever. Werner (p. 986) discusses this outbreak, which resembled typical trench fever.

Dyer (p. 986) gives a description of the properties of the virus, isolated from *Dermacentor andersoni*, which resembles that of Australian Q fever. There is complete cross immunity between the two, but none with the Rocky Mountain fever virus. *Proteus X* is not agglutinated.

Louse- and flea-borne.—In Algeria Fournier (p. 988) shows that small mild epidemics occurred in rural areas in 1937, especially in winter and spring. The usual methods of prevention, without vaccination, proved successful. Meunier (p. 988) gives figures for recent years. In Morocco (p. 988) there are small outbreaks every year but so long as the people are scattered this is of little moment. During famine the population is attracted towards towns and collections of natives live round the walls of cities. This overcrowding favours epidemic spread. Drought and famine must be overcome by irrigation and improvement in economic conditions, and other usual methods, including vaccination, must be employed.

Scaffidi (p. 989) describes the typhus, probably classical louse-borne type, seen in Northern Ethiopia. It varies considerably in

severity but he thinks that in the natives there is a special resistance of the cutaneous vascular system. Full serological investigation was not possible. In the treatment of typhus in Abyssinia, GIUNTA and D'IGNAZIO (p. 989) give intravenous mercurochrome with ascorbic acid and vitamin B_1, glucose and cardiac stimulants.

THOMPSON (p. 990) describes an outbreak of (probably) louse-borne typhus in a member of the Viceregal bodyguard in India and in contacts. The first infection appears to have been contracted in Kashmir. CLEAVE (p. 990) reports a case positive to *Proteus OX19* whose infection was contracted in Shanghai.

JORDAN and FLETCHER (p. 990) find that an *impure* cresylic product is lethal to lice if ammonia is added.

DYER (p. 991) shows that there has been a considerable increase in the numbers of cases and distribution of endemic typhus reported in the United States in recent years. Practically all the small rodents examined are susceptible. KEMP (p. 991) shows that in Texas endemic typhus is usually contracted at places of work or in rat-infested houses. He considers it to be distinct from Brill's disease which Zinsser has shown to be caused by the classical virus.

LORANDO and PAPANASTASSIOU (p. 991) quote two patients, mother and son, with endemic typhus. The rash in the son was profuse but none occurred in the mother. LORANDO (p. 992) reports double orchitis in a case of endemic typhus. BRUNEAU (p. 992) describes a case of endemic typhus.

ZIA *et al.* (p. 992) describe the techniques for culture and staining of murine Rickettsia which were successful in experiments on 42 animals. ANIGSTEIN and LAWKOWICZ (p. 993) cultivated 8 strains of murine virus from infected rats. Six of these were Rickettsial in appearance and serological properties, the other two were more like Proteus. Living and formol-killed Rickettsia produced immunity to murine typhus virus.

Murine typhus virus injected into the peritoneum of white mice produces paralysis and LAIGRET and DURAND (p. 993) compare it with the neurotropic virus of yellow fever. BRUNEAU (p. 993) found that murine typhus virus did not produce fever when injected into Tonkingese patients suffering from nervous diseases. He concludes that many cases in the natives pass undetected.

VIOLLE (p. 994) succeeded in infecting rats by allowing them to feed on material contaminated with urine from infected rats and human patients.

GIROUD and PANTHIER (p. 994) show how the virus of murine typhus gains in virulence for white mice on passage through these animals.

RUIZ CASTAÑEDA (p. 994) describes a mild form of typhus, probably murine, endemic in Zacoalco. *Proteus X19* was agglutinated in one case.

IWATA (p. 995) classes the strains found in Manchoukuo as murine, intermediate and epidemic and shows how the clinical features correspond with the biological characters.

LÉPINE (p. 995) and FERNANDO (p. 996) describe clinical cases of typhus.

Tick-borne.—GEAR (p. 996) finds that in South Africa the agglutination of Proteus *OX19* rises to high titre in louse-borne typhus; that of *OXK* is irregular. The disease is therefore in the same group as classical European typhus. In guineapigs orchitis is rare. He recognizes two forms of tick typhus, one mild with constant primary

sore and contracted from veldt ticks, the other severe with occasional *tache noire* and contracted from dog ticks. In serological reactions, morphology and distribution of the Rickettsia, South African tick typhus resembles Rocky Mountain and boutonneuse fever. He (p. 997) considers that the severe type of tick typhus occurs in older people and is transmitted by adult *Haemaphysalis leachi* from domestic dogs in towns.

Blood from a case of spotted fever reported by FITZPATRICK (p. 998) produced fever and orchitis in guineapigs, which were later found to be immune to a virulent Rocky Mountain fever strain.

BAUERSFELD (p. 998) reports an unusual and fatal case of Rocky Mountain fever apparently contracted in Florida.

PHILIP and DIAS (p. 998) were unable to transmit the virus of Rocky Mountain fever through various Reduviid bugs.

DE MAGALHÃES (p. 998) found the virus of Miñas Geraes fever in rabbits, opossums, ticks and bed-bugs. The virus passes through the placenta of Rhesus monkeys, but not through shaved skin ; it rarely passes an L5 filter candle but is cultivable on the chorio-allantoid membrane of fowl embryo.

DE MAGALHÃES (p. 999) notes that although clinical diagnosis is enough in typical cases of Miñas Geraes fever, in mild cases further means must be employed.

Mite-borne.—BRUNEAU and CHAPUIS (p. 999) describe a case positive to *Proteus OXK* ; blood inoculated into the eye of a rabbit gave iridocyclitis with keratitis and continued fever, the typical tsutsugamushi reaction.

GUNTHER and SCHROEDER (p. 1000) show that 46 Europeans have been infected with the *Proteus OXK* type of fever in a district of New Guinea since 1934. The bush is heavily infested with mites. WIJERAMA (p. 1000) reports two cases of *Proteus OXK* type of fever in Ceylon. In the Pescadores KAWAMURA and YAMAMIYA (p. 1000) show that 32 cases of *Proteus OXK* type of fever have been investigated.

Vaccination.—In the troops in Morocco, LAURENS *et al.* (p. 1000) found that cases of typhus occurred even among the vaccinated and believe this to be due to the fact that the virulence of the vaccine cannot be maintained constant and that if kept too long it loses its efficiency. They suggest the use of dried excreta of fleas.

GAUD (p. 1001) reports satisfactory results from widespread vaccination by the method of Blanc during an epidemic in Morocco. MARIANI (p. 1001) shows that in 13,000 vaccinations by the method of Weigl in Ethiopia, only a few persons showed severe reactions. A few cases of typhus were seen in the vaccinated but these were usually mild.

ZINSSER *et al.* (p. 1002) found that animals vaccinated with phenol- or formol-killed agar culture of Rickettsia were immune to test doses. VEINTEMILLAS (p. 1002) used Zinsser's formol vaccine and found that it protected human volunteers against infection with 2,000 minimum infective doses. One dose of the vaccine protected against murine virus, but 3 or 4 doses did so against classical virus also. It is proposed to use this vaccine on a large scale in Bolivia. *C. W.*

SARKAR (S. K.). **A Case of Typhus Fever.**—*Indian Med. Gaz.* 1939. Apr. Vol. 74. No. 4. pp. 223–224.

A case of fever in a European in which the Widal reaction was negative but the Weil-Felix was positive for *Proteus OX2* with a

rising titre and was negative for *OX19* and *OXK*. The patient was convinced that he had not been recently bitten by a tick, flea or mite. This was the first and only case seen in this district for some years.

D. Harvey.

GUINDY : REPORT OF THE KING INSTITUTE OF PREVENTIVE MEDICINE FOR YEAR ENDING 30TH SEPTEMBER 1937 [SHORTT (H. E.), Director.] pp. 28–31.—**Tick Typhus Enquiry.**

Twelve cases of typhus were notified in the Madras Presidency in 1936. Accordingly all sera sent to the Institute and found negative for the Widal reaction were tested for the Weil-Felix reaction. Four hundred and thirty-eight sera have so far been tested ; titres of 1/200 and above were taken as positive. Sixty-five sera gave such positive reactions, 42 with *Proteus OX19*, 16 with *OXK* and 7 with *OX2*. Eight cases of simple continued fever were examined but it was not possible to isolate a typhus virus from any of these nor from 4 cases which had been definitely diagnosed as typhus. Numerous rats, bandicoots and squirrels were examined but no virus was isolated from any of these ; nor from lice, mites, fleas or ticks removed from these animals. *D. H.*

BLANC (Georges) & NOURY (M.). Etude sur l'immunité cutanée du lapin vis-à-vis des virus de typhus murin et de fièvre boutonneuse. [**A Study of the Cutaneous Immunity of the Rabbit against the Viruses of Murine Typhus and Boutonneuse Fever.**]—*C. R. Soc. Biol.* 1939. Vol. 130. No. 3. pp. 234–236.

If the virus is inoculated into the skin of the left flank of a rabbit a local reaction is produced at the site of the needle puncture and also a general infection results. If 9 to 15 days later the animal is inoculated intradermally in both flanks there is no reaction in the left side but a slight reaction is noted in the other flank. After 15 days a positive reaction occurs at both sites of inoculation and after 25 days the reaction is always positive and is just as marked as the original reaction, especially on the side which did not receive the primary injection. The virus can always be isolated from the local lesions.

When a third inoculation is made 16 days after the second there is a local reaction on both sides, and a fourth inoculation 42 days later gives a similar result.

If the rabbits have been previously inoculated intraperitoneally the results of intradermal injection are similar to those described above. The virus of boutonneuse fever gives the same reactions as the virus of murine typhus.

From a study of the above results it is obvious that general and visceral immunity are not correlated with cutaneous immunity.

D. H.

GIROUD (Paul). Variations des réactions cutanées locales à la suite d'inoculations successives de Rickettsies du typhus murin. [**Variations in the Local Skin Reactions following Successive Inoculations of the Rickettsia of Murine Typhus.**]—*C. R. Soc. Biol.* 1939. Vol. 130. No. 3. pp. 230–233.

Rabbits were used in these experiments. Normally the first intradermal injection of 0·2 cc. of virus produces a local reaction which runs

its course from the 4th to the 10th day and reaches its maximum about the 6th day. Dilutions of 1/25, 1/125, 1/250, 1/500 and 1/1,000 were employed. If the second intradermal inoculation is made on the 3rd day after the first injection no reaction is produced even with the 1/25 dilution ; if the second injection is made on the seventh day a slight reaction appears on the second day ; but if the second injection is made after 18 days there is a marked nodular reaction even with a dilution of 1/1,000. This reaction is more nodular and greater than the primary reaction. In animals inoculated intraperitoneally and then 12 days later given an intradermal injection a marked local reaction results. Thus general immunity is not shared by the skin.

Boutonneuse virus gives the same reaction and the virus is present in the local skin lesion. The local skin reaction is most marked in animals which are least susceptible to general infection with the virus and in susceptible animals the skin reaction is slight. *D. H.*

Giroud (P.). Réactions d'immunité locale provoquées par des inoculations répétées de rickettsies. [**Reactions of Local Immunity provoked by Repeated Inoculations of Rickettsia.**]—*Bull. Soc. Path. Exot.* 1939. Mar. 8. Vol. 32. No. 3. pp. 251–258. With 2 figs.

The inoculum used was emulsion of guineapig tunica rich in Rickettsia. If the second injection is given soon after the first there is practically no reaction in the skin ; but if later a marked nodular reaction is seen. General immunity does not extend to the skin. This general immunity is shown by the fact that there is no reaction to intraperitoneal inoculation after repeated intradermal injections which continue to show local reactions. *D. H.*

Raynal (J.) & Fournier (J.). Sur les virus de typhus exanthématique isolés à Changhaï en 1938. [**On the Typhus Viruses isolated in Shanghai in 1938.**]—*Bull. Soc. Path. Exot.* 1939. May 10. Vol. 32. No. 5. pp. 525–536. With 6 charts.

Three strains of typhus virus were examined : LB isolated on the 4th day of fever in a European who recovered ; OYZ isolated on the 17th day from a Chinese who died ; M a virus isolated from local rats.

The results of tests on guineapigs, rats and rabbits showed that virus M was a true murine virus and OYZ and LB were intermediate. OYZ was nearer to the classical virus and LB to the murine virus.

D. H.

Raynal (J.) & Fournier (J.). Le signe de Neill-Mooser dans le typhus expérimental. [**The Neill-Mooser Sign in Experimental Typhus.**]—*Bull. Soc. Path. Exot.* 1939. Feb. 8. Vol. 32. No. 2. pp. 188–195. [29 refs.]

Describes the Neill-Mooser reaction in guineapigs after injection of infective blood from man or other experimental animals such as rats or mice. In the latter case the reaction (orchitis) may be caused by *Bact. paratyphosum B* which gives a very similar appearance but can be differentiated by the absence of Rickettsia and also the positive blood culture. The spirochaete of rat-bite fever may also cause confusion but the reaction appears later than with typhus virus and the condition is harder, more painful and lasts longer ; also a dose of novarsenobenzol cures the fever and orchitis in rat-bite fever but has

no effect in typhus. That the condition is due to typhus virus can be proved by a study of the temperature chart, immunity and agglutination tests. The older and heavier guineapigs give the most marked reaction, which varies according to the resistance of the animal and the origin of the virus—blood, brain, etc. The scrotal reaction may appear whatever the source of the virus, but is most common with that of murine typhus and occurs only rarely with that of historic typhus.

D. H.

Nicolle (P.) & Simons (H.). Accélération de la sédimentation des hématies dans le typhus murin expérimental du cobaye. [**Acceleration of the Sedimentation of the Red Blood Cells in Experimental Typhus in the Guineapig.**]—*Bull. Soc. Path. Exot.* 1938. Dec. 14. Vol. 31. No. 10. pp. 937–946. With 2 graphs.

Fifty guineapigs were infected and the reaction tested and compared with 90 control animals.

Conclusions.—In experimental typhus the acceleration of the rate of sedimentation of the red cells is considerable. It commences early in the illness and reaches its maximum on the 8th day, decreases rapidly from the 9th to the 12th and more slowly from 13th to 24th day. Two months after infection it disappears completely. When animals which have recovered are reinoculated with the virus acceleration does not occur again.

It is necessary to exclude bacterial infections as these may also produce the same effects. *D. H.*

Lestoquard (F.). L'iode dans la coloration des Rickettsia. [**Iodine in the Staining of Rickettsia.**]—*Bull. Soc. Path. Exot.* 1939. May 10. Vol. 32. No. 5. pp. 466–467.

This method was first suggested in 1926. Films are fixed in iodized alcohol 98 parts alcohol, 2 parts iodine, for 8 to 10 minutes. Wash in pure alcohol to remove the iodine, then stain with Giemsa's stain. This method has been used by the author in his work on *Rickettsia canis*. *D. H.*

Giroud (P.) & Panthier (R.). La réaction au lugol sur les cellules parasitées par les rickettsies. [**The Reaction to Lugol of Cells containing Rickettsia.**]—*Bull. Soc. Path. Exot.* 1939. Jan. 11. Vol. 32. No. 1. pp. 14–17.

This reaction has been described in trachoma, and the authors tried out the same technique using films made from infected tissues of guineapigs. The Mooser cells in the films did not give any reaction. Some cells gave reactions but these did not contain Rickettsia. It is suggested that the reaction is due to staining of glycogen in the cells by lugol. [Presumably this refers to Lugol's solution.] *D. H.*

Cox (Herald R.). **Use of Yolk Sac of Developing Chick Embryo as Medium for Growing Rickettsiae of Rocky Mountain Spotted Fever and Typhus Groups.**—*Public Health Rep.* 1938. Dec. 23. Vol. 53. No. 51. pp. 2241–2247.

Inoculation was made into the embryonic membrane enclosing the yolk mass.

The inoculum was :

Rocky Mountain fever virus	...	blood of guineapig
Endemic typhus virus ...	...	testicular washings
Boutonneuse fever virus ...	...	,, ,,
Brazilian spotted fever virus	...	blood of guineapig
European typhus ...	...	,, ,,

It was found possible to pass all these viruses in series from egg to egg ; in many instances the embryo was killed by the infection by the 3rd or 4th day. It was also found that the virulence of the inoculum for guineapigs was increased by passage in this manner ; it was from 100 to 1,000 times more infective than the same virus from mammalian tissues and was comparable to the virulence in the tissues of the tick.

D. H.

Pigoury (L.) & Bernard (M.). Existence de *Rickettsia canis* dans le Proche-Orient. [**Existence of *Rickettsia canis* in the Near East.**]—*Bull. Soc. Path. Exot.* 1939. Jan. 11. Vol. 32. No. 1. p. 19.

A number of sick dogs were examined, films made from lung puncture were stained but no *R. canis* were noted. Forty-six dogs from the local pound were examined similarly and in one, 6 months old, numerous Rickettsia were noted in the mononuclear cells in a film from a lung puncture.

The stained film was sent to MM. Donatien and Lestoquard and they definitely diagnosed the parasite as *R. canis*.

This is the first time this parasite has been noted in the Eastern Mediterranean (Beirut). *D. H.*

Herzig (Anna). Eine neue Rickettsia-Spezies der Laus, der Erreger einer spontan aufgetretenen epidemischen Erkrankung des Menschen. [**A New Species of Rickettsia of the Louse the Cause of a Spontaneous Epidemic of Fever in Man.**]—*Zent. f. Bakt.* I. Abt. Orig. 1939. Mar. 30. Vol. 143. No. 5/6. pp. 299-302.

Forty to fifty people in Weigl's laboratory feed voluntarily the normal lice which are bred in large numbers for purposes of preparing anti-typhus vaccine. Approximately 300,000 lice are fed daily.

In April 1938, two of these men developed fever with pains in head and back ; the fever only lasted 2 or 3 days but was followed by relapse. Later the greater number of the men became ill with the same symptoms and a large percentage of the lice showed Rickettsia infection. These organisms were extracellular and resembled *R. pediculi*. Rickettsia could be seen in the blood of the patients in large numbers. It was possible to reproduce the disease in volunteers by injecting subcutaneously the gut contents of infected lice and also by feeding lice on normal men. The incubation period was about 3 days. The Rickettsia can be passaged from louse to louse but soon loses its pathogenicity for man. No infection could be produced in any of the usual experimental animals.

All the characters of this Rickettsia remind one of *R. weigli* and also of *R. quintana* the cause of trench fever. Further experiment will be necessary to decide whether these three Rickettsia types are actually one and the same or are only morphologically similar.

The author is inclined to the view that these types are distinct from one another and differ just as do different strains and types of dysentery bacilli which produce different types of clinical dysentery.

D. H.

WERNER (H.). Neuere Ergebnisse der Fünftagefieberforschung. [**Recent Investigations on Five Day Fever.**]—*Deut. Med. Woch.* 1939. Feb. 3. Vol. 65. No. 5. pp. 174–176.

During the years of the Great War there were large epidemics of trench fever, also many cases of the disease were missed and classified as simple continued fever, slight rheumatism or influenza.

At the end of the war the disease disappeared as suddenly as it had come. For this reason it was not possible to carry out research on any large scale since the war. Yet the disease has been reported from time to time in Poland, Galicia and in Japan. In 1938 Professor WEIGL wrote to the author and stated that in 1934 there were 18 cases of clinically typical trench fever among the personnel in his laboratory. About 40 persons were used to feed the stock lice and 18 of them developed the fever and the Rickettsia found in the lice were similar to *R. quintana.*

The symptoms of the patients corresponded with those of trench fever seen during the Great War. All these people had been previously vaccinated with Weigl anti-typhus vaccine.

Inoculation of clean lice was successful and the Rickettsia in the lice were extracellular; normal people were infected from these lice. The author refers to the work of OGATA in Japan where cases of five day fever have occurred from time to time and this observer has succeeded in isolating the virus from the blood of patients by intratesticular inoculation in rabbits—pure cultures have been thus obtained and the disease has been produced in man by inoculation of these cultures. In 1927 Werner suggested the use of the virus of trench fever in the treatment of neurosyphilis and it has been so used recently by Ogata in Japan.

D. H.

DYER (R. E.). **Similarity of Australian "Q" Fever and a Disease caused by an Infectious Agent isolated from Ticks in Montana.**—*Public Health Rep.* 1939. July 7. Vol. 54. No. 27. pp. 1229–1237. With 6 figs. [12 refs.] [Summary appears also in *Bulletin of Hygiene.*]

In 1935 DAVIS and COX obtained from *Dermacentor andersoni* a virus (X) which would pass through Berkefeld N and W filters, which held back bacteria and the viruses of typhus and Rocky Mountain spotted fever. The virus survives in and is transmissible by the Dermacentor in nymphal and adult stages, if ingested in the larval stage, and it survives through the eggs to the larva. It does not produce agglutinins for *Proteus X* strains, nor did it infect *M. rhesus.* In guineapigs it produces fever for 2–8 days after an incubation of 4–6 days; there is no scrotal reaction. Guineapigs recovered from Q fever virus were found immune to the new virus of Davis and Cox. *Rickettsia burneti* is found in spleens of mice infected with Q fever,

and workers in Australia have found *M. rhesus*, guineapigs, white mice, several native rodents, the bandicoot (*Isoodon macrourus*) susceptible. In guineapigs Q virus produces a febrile reaction for 4–6 days after an incubation ranging between 2 and 18 days; again, there is no scrotal reaction.

As stated above guineapigs recovered from A fever were immune to inoculation with X virus. Other cross immunity tests were carried out with two strains of typhus fever, one endemic and one epidemic, with two of Rocky Mountain spotted fever, one avirulent strain from Montana and a milder one from Maryland. Figures in the text show that there is no cross immunity between the X strain and the typhus and spotted fever strains, nor between the A fever and spotted fever strains, but, as stated above, there is complete cross immunity between X and Q fever strains. Other figures demonstrate the results of agglutination and protection tests with the different sera. Briefly, as regards the subject of this article, definite protection against X virus was given by X serum and Q fever serum, but none by spotted fever serum. The author sums up the points of similarity and difference as follows :—

" *Epidemiology.*—' Q ' fever has been recognized principally in persons associated with animals, which suggests infection from direct contact with infected animal tissues or with animal parasites.

" The epidemiology of the Montana infection is unknown, but the presence of the virus in ticks suggests that human infections may be found in rural areas.

" *Clinical.*—The one recognized human infection with the Montana virus was very similar to the published descriptions of the Australian ' Q ' fever cases.

" *Susceptibility of animals.*—As far as work has been carried out, the only point of difference in susceptibility of animals to the two infections is the failure of the American workers to find the monkey susceptible, in contrast to the success in infecting this animal in Australia."

" *Serology in man and animals.*—Neither disease has been found to produce agglutinins for *Proteus X* strains. It should not be forgotten that the opportunity to study this point in human beings in this country has been limited to one case [an accidental infection of a laboratory worker in Montana].

" *Reactions in guinea pigs.*—The clinical pictures in guinea pigs, as described in the literature, are similar, with the exception that the Montana infection has been reported to produce a definite local skin reaction following subcutaneous inoculation, while the Australian workers state that no particular local reaction follows subcutaneous inoculation. A comparison of the two strains in this laboratory shows that the ' Q ' fever strain produces general reactions in guinea pigs which, although similar to those produced by the Montana virus, are milder. This fact may be explained by the attenuation of the ' Q ' virus during transit to this country. Rickettsiae have not been observed in guinea pigs with ' Q ' fever, while they are present in abundance in guinea pigs infected with the Montana virus.

" *Cross immunity tests.*—These tests are identical, with the exception that epidemic typhus, and, to a lesser extent, endemic typhus apparently produce more immunity to ' Q ' fever than to the Montana virus.

" *Agglutination of rickettsia.*—In one well-controlled test the serum from one recovered case of the Montana infection gave results identical with one serum from a recovered case of ' Q ' fever when tested with a suspension of *Rickettsia burneti* prepared in Australia.

" *Protection tests.*—As far as these tests have been tried no immunological difference has been noted between the virus of ' Q ' fever and that isolated from Montana ticks."

[See this *Bulletin*, 1938, Vol. 35, pp. 62–64, 835; 1939, Vol. 36, p. 479 and *Bull. of Hyg.*, 1938, Vol. 13, pp. 122, 123, 673.]

H. H. S.

Fournier. L'évolution épidémiologique du typhus exanthématique observée en Algérie au cours de l'année 1937. [**The Epidemiology of Typhus in Algeria during the Year 1937.**]—*Bull. Office Internat. d'Hyg. Publique.* 1938. Dec. Vol. 30. No. 12. pp. 2759–2766.

The author's conclusions may be summarized as follows :—

1. Typhus has occurred in small epidemics of mild cases in the rural areas but has spared the towns.
2. This distribution of cases depends on the economic situation of the areas in question.
3. Most of the cases occurred in the winter and spring and followed a normal clinical course usually of a mild character.
4. The usual methods of prevention, isolation, disinfection, sanitary supervision, etc., proved successful. Contacts were given convalescent serum as a precaution but vaccination was not carried out on a large scale.

D. H.

Meunier (R.). Quelques données numériques concernant le typhus exanthématique en Afrique du Nord et particulièrement en Algérie. [**Numerical Records concerning Typhus in North Africa and Specially in Algeria.**]—*Bull. Office Internat. d'Hyg. Publique.* 1938. Dec. Vol. 30. No. 12. pp. 2767–2771. With 3 charts.

A statistical paper.

In 1937 there were 3,299 cases of typhus reported in Algeria, 3,804 in Tunisia and 1,822 in Morocco, a total of 8,925 in French North Africa.

The last big epidemic of typhus was in 1928 when there were 4,760 cases; in 1929 only 448 cases were reported. *D. H.*

Bulletin de l'Institut d'Hygiène du Maroc. 1938. Jan.–June. No. 1–2. pp. 51–65.—La prophylaxie du typhus au Maroc. [**Prophylaxis of Typhus in Morocco.**]

The epidemic of typhus in Morocco ceased in August 1938 and during the interim the following suggestions were made with a view to prevention of recurrence.

The main cause of the previous epidemic was the almost complete failure of the crops of the peasants in the country surrounding the cities of Marakeesh and Casablanca; this resulted in the migration of these destitute and starving people into the cities and the formation of "tin" towns around the walls of the cities. Every year in Morocco there are small outbreaks of typhus among the local inhabitants, but as long as these people are scattered about in small villages and camps no large epidemic occurs, but in 1937 all or practically all these people were forced by the menace of starvation to seek shelter and food in the cities with the result that all were crowded together in slums and institutions, and an epidemic of typhus resulted. One of the chief measures of prevention must be improvement of the economic condition of the people by ensuring sufficient crops to feed them in their own villages and farms, and thus spreading them out once more

over the country districts. The main cause of the failure of crops is drought and this must be overcome by irrigation measures on a large scale. Also the usual medical and sanitary measures must be energetically carried out: notification of all deaths and investigation of the causes, cleansing of towns, buildings and streets, arrest of beggars and segregation in camps and institutions. Early detection of cases of typhus and isolation of these; lousing of people, their bedding and houses; isolation of contacts; vaccination of contacts and the general population by bile treated vaccines, with propaganda and education of the people, are other measures advised. [See GAUD below, p. 1001.] *D. H.*

DUPOUY. Renseignements sur le typhus exanthématique au Maroc et à Casablanca. [**Notes on the Epidemic of Typhus in Morocco and Casablanca.**]—*Arch. Méd. et Pharm. Nav.* 1938. July–Aug.–Sept. Vol. 128. No. 3. pp. 706–733. With 1 map.

Notes on the epidemic of typhus in Morocco and Casablanca with special reference to the naval personnel. *D. H.*

SCAFFIDI (Vittorio), Jr. Il dermotifo tra gli indigeni a cute nera del Nord-Etiopia. (Particolarità sintomatiche e cenni epidemiologici.) [**Typhus in the Natives of Northern Ethiopia.**]—Reprinted from *Giorn. Clin. Med.* 1937. Vol. 18. No. 16. 12 pp. With 5 charts. [16 refs.]

This is a detailed account of the clinical features of typhus as seen in 30 natives, 19 from a military prison in Asmara, the remainder from various parts of the territory. In black skins the rash shows variations in intensity, but is often only a transient macular exanthem. No rash may be seen, and the author inclines to the opinion that in natives there is a special resistance in the cutaneous vascular system, since haemorrhagic lesions are found post-mortem in the visceral capillaries but not in those of the skin.

He draws attention to a characteristic fall in temperature on the second day of fever and shows that termination is either by crisis or lysis. The type of disease he has seen conforms to classical exanthematic typhus because, (1) Rickettsia were found in lice from the patients, (2) the outbreak in the military prison was rapidly stamped out by measures taken against lice, and (3) inoculation of blood into guineapigs produced fever but no orchitis, and into rats did not produce disease. The Weil-Felix reaction was positive in titres of 1/200 to 1/3,500 in those cases in which it was performed, but laboratory facilities were not adequate for full serological investigation.

The disease varied in severity from a mild form to forms with severe complications, bronchopneumonia, purulent parotitis, gangrene of the toes and death. Treatment by subcutaneous injections of 0·04 gm. salicylate of mercury on alternate days was employed in some cases, but without apparent benefit. *C. W.*

GIUNTA (Beppino) & D'IGNAZIO (Camillo). La terapia del dermotifo. [**The Treatment of Typhus.**]—*Rass. Sanitaria dell' A.O.I.* Addis Ababa. 1939. July 9. Vol. 1. No. 2. pp. 11–16. English summary (5 lines).

Although it is not possible to determine how far the haemorrhagic lesions characteristic of typhus are caused by lack of vitamin C, it is

legitimate, by analogy with other infective diseases, to consider that this hypovitaminosis may play a considerable part. It is also possible to consider that B hypovitaminosis may be concerned with the vascular and perineural lesions. On these suppositions the authors attempted various combinations of vitamin treatment combined with other methods. They had previously found that mercurochrome and strychnine had proved useful and the treatment now advocated, after an experience of 40 cases, consists of intravenous administration of 2 cc. mercurochrome (1 per cent.) daily for 5 or 6 days ; intravenous injections of 25 cgm. ascorbic acid (as Cebion, etc.) twice to four times daily ; administration of 10 mgm. vitamin B_1 (Betaxin intramuscularly) twice daily, with lumbar puncture if the c.s.f. is under pressure ; and symptomatic treatment with subcutaneous glucose and ordinary cardiac stimulants.

Protocols of these experiments will be published later but in the meantime the authors state that excellent results have been obtained in this way in Addis Ababa. [See also SCAFFIDI above.] *C. W.*

THOMPSON (T. O.). **Fevers of the Typhus Group in Northern India.**—*Jl. Roy. Army Med. Corps.* 1939. Apr. Vol. 72. No. 4. pp. 267–269.

This note refers to an outbreak of fever in the Viceregal bodyguard at Dehra Dun in the hot weather of 1929. There were 7 cases with 4 deaths ; the medical attendants attributed the fever to bites of ticks incurred while grazing horses in the neighbourhood. On investigation, however, it was discovered that the first patient was taken ill 4 days after he had returned from leave at his home in Kashmir State on one of the main trade routes through the hills [see BLEWITT, this *Bulletin*, 1939, Vol. 36, p. 461]. He had attended a festival 14 days before he developed fever ; 10 to 12 days later 3 contacts developed fever and 3 more 10 days later. One case only was seen by the author. He had very high fever and was critically ill ; body lice were seen crawling away over the bedclothes ; the patient died a few hours later. The inference is that the cases were true louse-borne typhus and not tick typhus. *D. H.*

CLEAVE (T. L.). **A Case of Typhus Fever.**—*Jl. Roy. Nav. Med. Serv.* 1939. Jan. Vol. 25. No. 1. pp. 72–73. With 1 chart.

A case of typhus contracted in Shanghai and detected during transit to Hongkong. The Weil-Felix reaction was positive 1/640 for *Proteus OX19*. Two guineapigs inoculated with blood taken from the case both developed fever and a second guineapig inoculated from these also became infected. This work was carried out in hospital in Hongkong. *D. H.*

JORDAN (J. H.) & FLETCHER (A. E.). **A Note on Disinfestation with Particular Reference to Typhus.**—*Chinese Med. Jl.* 1938. July. Vol. 54. No. 1. pp. 71–72.

In a letter to the *Tropical Diseases Bulletin* the authors point out that in experiments it was found that ordinary cresylic acid or refined products such as Lysol, Izal, etc., were only slightly toxic to lice even with the addition of ammonia. An *impure* cresylic product containing

pyridine, quinoline and naphthalene, however, had a lethal effect on lice if ammonia was added. This product was cheaper than the pure product, and the mixture with ammonia was useful in bug infestation. They suggest that quinoline might prove worthy of investigation in regard to its use in disinfestation. [See this *Bulletin*, 1939, Vol. 36, p. 472.] *C. W.*

Dyer (R. E.). Les maladies à Rickettsia aux États-Unis. [**Rickettsial Diseases in the United States.**]—*Bull. Office Internat. d'Hyg. Publique.* 1938. Dec. Vol. 30. No. 12. pp. 2772–2774.

Two Rickettsial diseases are endemic in the United States, endemic typhus and Rocky Mountain spotted fever. In 1923 only 50 cases of endemic typhus were reported and all of these were in the coast towns of the south-eastern States. Since that date there has been a gradual increase in the numbers reported and it has spread from the towns to the country districts; 1,308 cases were reported in 1934 and in 1937 in Georgia alone more than 1,000 cases were reported. Practically all the small rodents, natives of the southern States, which have been examined have been proved to be susceptible to the virus. Spotted fever also is now no longer confined to the Rocky Mountains, but is also found in the eastern States in Canada, British Columbia and in Brazil. *D. H.*

Kemp (Hardy A.). **Endemic Typhus Fever in Texas. An Epidemiological and Clinical Comparison with Forms of Typhus seen elsewhere.**—*Amer. Jl. Trop. Med.* 1939. Mar. Vol. 19. No. 2. pp. 109–129. [18 refs.]

In Texas the disease attacks young male adults and in the temperate regions the cases occur in August and September. Most of the cases are in middle-class people who contract the disease not in their homes, but at their work in shop or store. On the other hand poor whites contract the disease in rat-infested houses.

The clinical description shows that the disease is similar to that met with elsewhere but the author insists that the disease is distinct from Brill's disease which Zinsser has shown is caused by the classical virus. *D. H.*

Lorando (N.) & Papanastassiou (E.). Typhus endémique murin sans exanthème. [**Endemic Typhus without Rash.**]—*Bull. Soc. Path. Exot.* 1939. Feb. 8. Vol. 32. No. 2. pp. 122–124.

Two cases of fever occurred at the same time in the same house, a man aged 45 and his mother aged 62. Both cases were typical and gave strong positive Weil-Felix reactions. A virus was isolated from the blood and produced fever and orchitis.

The two cases were similar except that in the older patient no sign of rash could be discovered although careful examination was made daily. In the case of the son the rash was profuse and typical.

D. H.

Lorando (N.). Périorchite chez un malade atteint de typhus murin. [**Periorchitis in a Case of Murine Typhus.**]—*Bull. Soc. Path. Exot.* 1939. Jan. 11. Vol. 32. No. 1. pp. 17–18.

A case of fever with profuse typhus rash and Weil-Felix reaction positive for *Proteus OX19* in a dilution of 1/500. Just as the fever ceased on the 20th day a double orchitis developed with pain and tenderness ; the condition lasted for one week. *D. H.*

Bruneau (J.). Un cas de typhus murin, isolement et identification. [**A Case of Murine Typhus ; Isolation and Identification of the Virus.**]—*Rev. Méd. Française d'Extrême-Orient.* 1938. Aug.–Sept. Vol. 20. No. 7. pp. 902–906. With 18 charts.

A case of fever in a European with typical rash and other symptoms. The Weil-Felix reaction was positive for *Proteus OX19.* Blood was taken during the fever and inoculated into guineapigs ; these reacted with fever and orchitis. Rickettsia were present in smears made from the inflamed tunica. The virus was passaged for 13 generations in guineapigs ; monkeys were also infected and when tested later were found to be immune to the viruses of murine typhus and true typhus.

D. H.

Zia (Samuel H.), Liu (P. Y.) & Pang (K. H.). **Isolation of Typhus Rickettsia from Infected Animals on Zinsser Agar-Tissue Media.**—*Chinese Med. Jl.* 1938. Dec. Vol. 54. No. 6. pp. 547–558. With 1 fig.

The agar for culture of Rickettsia was prepared according to the directions of Zinsser.

Take 150 cc. double distilled water with 3·2 gm. of sod. chlor. ; add

2 per cent. pot. chlor.	...	4 cc.
2 per cent. calc. chlor.	...	4 cc.
1 per cent. mag. chlor.	...	4 cc.
5 per cent. sod. bicarb.	...	4 cc.
0·5 per cent. pot. ac. phos.	...	4 cc.

Add 0·4 gm. glucose

Horse or human serum	...	125 cc., filter
Tyrode double strength	...	100 cc.

Boil 4 per cent. agar. To 100 cc. agar add 110 cc. of filtered Tyrode+serum. Add 10 cc. 0·02 per cent. phen. red. Tube agar and slope. 7·8 *p*H.

Three strains of murine Rickettsia were used, a Mexican strain, a strain from a local rat and from a local patient. Small pieces of tunica, removed aseptically from infected rats, were placed on the surface of the agar tubes and incubated at 37°C. Mouse embryo tissue was placed in the culture tubes and then transferred to new tubes of media for subcultures.

Smears were examined by staining as follows :—

3 minutes phosphate buffer, wash off.
4 minutes 0·25 per cent. basic fuchsin in distilled water.
Decolorize in 0·05 per cent. citric acid.
Counterstain 5 secs. with 0·25 per cent. aqueous methylene blue.

Successful cultures were obtained from 36 guineapigs and 6 rats and subcultures were carried for several generations. *D. H.*

Anigstein (Ludwik) & Lawkowicz (W.). **Researches on Strains of *Rickettsia* and *Proteus* cultivated from Experimental Typhus of the Murine Type.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1939. Feb. 28. Vol. 32. No. 5. pp. 605–613. With 3 figs. on 1 plate.

The culture medium employed in this research was Noguchi's semi-solid agar medium [see Lawkowicz below]. Two strains of murine typhus virus were employed, one obtained from Tunis and the other from Poland. Tests showed that these two strains were identical. The inoculum was the blood and brain of infected rats and guineapigs. The cultures were incubated at 35°C. for 5 days—8 strains were cultivated *in vitro* from these two strains, growth appeared after 4 days' incubation and consisted of masses of minute cocci or delicate rods. Six of these strains were definitely Rickettsial in appearance, were non-motile, did not ferment any sugars and later grew well on chocolate agar. Two strains were definite bacilli more like Proteus, these were motile and fermented carbohydrates with the production of acid and gas; they were non-indologenic. The 6 Rickettsia strains were agglutinated by immune rabbit serum to the same dilution, the Proteus strains were not so agglutinated. Guineapigs infected by injection of living Rickettsial cultures were later shown to be immune to the virus of murine typhus from passage animals. Formol killed Rickettsial cultures were used to vaccinate rats and immunity was produced against murine typhus virus. *D. H.*

Laigret (Jean) & Durand (Roger). Modification de l'activité pathogène de deux souches de virus typhiques murins entretenues par passages sur les souris blanches. [**Modification of the Pathogenic Activity of 2 Strains of Murine Typhus Virus during Passage in White Mice.**]—*C. R. Acad. Sci.* 1939. Feb. 27. Vol. 208. No. 9. pp. 673–675.

It has already been noted that the virus becomes much less virulent for guineapigs when passaged in white mice. But in the course of their investigation the authors found that if the peritoneal route is employed in the injection of the white mice paralysis is produced and these animals die. This condition is not produced either in rats or guineapigs by the passaged virus; although in one or two rats high fever was produced. It was found that in most cases there was a large amount of virus in the brains of the paralysed mice. This condition is compared with the neurotropic virus in mice in yellow fever. *D. H.*

Bruneau (J.). Contribution à l'étude expérimentale du typhus murin chez l'indigène tonkinois. [**A Contribution to the Experimental Study of Murine Typhus in the Natives of Tonking.**]—*Rev. Méd. Française d'Extrême-Orient.* 1938. Aug.–Sept. Vol. 20. No. 7. pp. 889–901. With 22 charts.

Three strains of virus were used in these experiments, one isolated from the brains of rats in Hanoi, one from a case of fever in Hanoi and the virus Tunis No. 1 sent from Morocco. These viruses were inoculated into Tonkingese suffering from nervous diseases and it was found that these people were not susceptible or only slightly so. Indeed unless the temperature had been taken at frequent intervals

no fever would have been detected and there were no symptoms of ill health. The viruses were of no use as producers of fever for treatment. The local viruses gave rise to less reaction than did the Tunis virus. The Weil-Felix reaction was positive in the cases and one inapparent case reacted to a dilution of 1/700. The conclusion is that many cases in the natives may pass undetected. *D. H.*

Wu (Chao-Jen) & Zia (Samuel H.). **Isolation of Murine Typhus Rickettsia from House Rats in Peiping.**—*Chinese Med. Jl.* 1939. Mar. Vol. 55. No. 3. pp. 231–238. With 1 chart.

Violle (H.). Expériences sur le virus exanthématique murin. Sensibilité du rat aux urines virulentes d'origine humaine et murine. [**Experiments with Murine Typhus Virus. The Susceptibility of the Rat to Infected Urine of Man and Rats.**]—*C. R. Soc. Biol.* 1938. Vol. 129. No. 33. pp. 984–986.

The author considers that rats were to be preferred to guineapigs for these experiments as they are more susceptible and are natural hosts of the virus.

Rats were fed on bread which had been soaked in the urine of rats infected with typhus and these animals became infected; positive results were also obtained with food contaminated with the urine of a human patient suffering from endemic typhus. The usual symptoms were observed in the infected rats.

This is a possible mode of infection from rat to rat in nature and man may also be infected by the consumption of food soiled by the urine of rats. It is also suggested that the diagnosis of cases might be aided by the feeding of rats on food contaminated with the urine of patients. [See also this *Bulletin*, 1938, Vol. 35, p. 782.] *D. H.*

Giroud (P.) & Panthier (R.). Comportement de la souris blanche à la suite de l'inoculation de virus typhique murin. [**Behaviour of White Mice following Inoculation of the Murine Typhus Virus.**]—*Bull. Soc. Path. Exot.* 1939. Apr. 4. Vol. 32. No. 4. pp. 404–409. With 5 figs.

If this virus is passaged in white mice it gains in virulence for these animals but loses virulence for guineapigs. If the virus is then passaged in guineapigs by the method of intraperitoneal injection of tunica washings the virulence is rapidly regained; but virulence is lost for white mice.

The Zinsser virus causes death in 90 per cent. of inoculated white mice in from 4 to 28 days. At the first passage death occurs from the 6th to the 14th day, at the 2nd passage 4th to 5th day and 3rd passage in 32 to 100 hours. After the 4th passage convulsions occur in the mice lasting about 15 seconds and death follows.

Rickettsia-like bodies were seen in the peritoneal exudate and also in the liver and spleen of the mice. *D. H.*

Ruiz Castañeda (M.). El tifo de Zacoalco. [**Typhus in Zacoalco.**]—*Medicina.* Mexico. 1939. Aug. 10. Vol. 19. No. 345. pp. 301–303. French summary (7 lines).

The author describes a mild form of typhus which is common in Zacoalco, Jalisco, and which had been considered clinically as a form

of typhoid fever. The fever is continuous for 10–12 days, without rash. Blood from one of the patients was sent to Guadalajara and there found to agglutinate *Proteus X19* and *Rickettsia prowazeki*, and to give the Neill-Mooser reaction in rats. Guineapigs immune to the strain L were immune to emulsions of the tunica of animals infected with the Zacoalco strain, and complete cross immunity was observed.

This strain therefore corresponds to the Mexican orchitic strain of exanthematic typhus, probably of murine origin. No body lice were found in the neighbourhood, but *P. capitis* was present. It is possible that transmission may occur from man to man through lice, but the origin of the infection from rodents and their ectoparasites cannot be excluded; investigation into its existence in rodents has not been undertaken. *C. W.*

Iwata (Shigeru). **Study on Typhus Fever. Of Types, Varieties and Clinical Symptoms of Typhus Fever in Manchuria.**—*Jl. Oriental Med.* 1939. May. Vol. 30. No. 5. pp. 193–220. With 1 map. [61 refs.]

The authors have examined a large number of strains of typhus virus isolated from cases in Manchoukuo. As a result of the effects of inoculation of these viruses into male guineapigs they found that they fall into 3 classes. The clinical picture, as seen in the Table below, corresponds with the laboratory findings :—

Virus

	Class I Murine	Class II Intermediate	Class III Epidemic
Scrotal reaction ...	Constant	Occasional	Absent
Mooser cells	Constant	Occasional	Absent
Clinical features			
Fever	Mild	More severe	Severe and critical
Mental symptoms ...	None	Occasional	Delirium
Rash	Macular	Petechial	Petechial
Fatal cases	None	Few	Many

In one epidemic all three types of virus were isolated from cases of fever. *D. H.*

Lépine (P.). A propos du typhus sans exanthème. [**Typhus Fever without Rash.**]—*Bull. Soc. Path. Exot.* 1939. Feb. 8. Vol. 32. No. 2. pp. 124–125.

A mild case of fever lasting 8 days. The Weil-Felix reaction was positive in a dilution of 1/1600. Blood was taken from the patient and inoculated into a guineapig, there was no orchitis but Rickettsia were found in smears from the exudate. This case occurred along with other cases which were typical and which all showed a rash. *D. H.*

FERNANDO (Cyril F.). **Tropical Typhus.**—*Jl. Ceylon Branch Brit. Med. Assoc.* 1938. Nov. Vol. 35. No. 6. pp. 463–466. With 1 chart.

Two cases of fever with rash. The Weil-Felix reaction was strongly positive for *Proteus OX19* in one case in a dilution of 1/5000; it was negative for *OXK*.

Guineapig inoculation was positive but Rickettsia could not be found in smear from the swollen tunica. *D. H.*

JULLIARD (J.) & HÉNAFF. Troubles du métabolisme hydrochloruré au cours des typhus épidémique et murin. Chloropexie vasculaire et système réticulo-endothélial. Considérations générales. [**Disturbances of Chloride Metabolism in Epidemic and Murine Typhus.**]—*Rev. Service Santé Milit.* 1939. Feb. Vol. 110. No. 2. pp. 197–266. With 10 charts. [Refs. in footnotes.]

GEAR (J. H. S.). **South African Typhus.**—*South African Jl. Med. Sci.* 1938. Oct. Vol. 3. No. 4. pp. 134–160. With 10 figs. [26 refs.]

A general review of the subject in the light of recent work by the author and his colleagues. PIJPER and others as the result of research were inclined to the view that the viruses of typhus fever in South Africa differed from the viruses of similar diseases elsewhere. [See this *Bulletin*, 1931, Vol. 28, p. 101; 1932, Vol. 29, p. 452, 779; 1933, Vol. 30, p. 909; 1935, Vol. 32, p. 154, 168, 561.] For instance, they found in the case of louse-borne typhus that the sera gave positive reactions only in low dilutions with *Proteus OX19* and also agglutinated *OX2* in similar or, in some cases, higher dilutions. For endemic typhus similar serological results were obtained. Tick bite fever as observed by the same observers was noted as being the mildest of the typhus-like fevers with a constant primary sore or ulcer, and was proved to be carried by larval ticks from veldt rodents to man. On the other hand RHODES [this *Bulletin*, 1935, Vol. 32, p. 561] in his area of South Africa found that the Weil-Felix reaction in louse-borne typhus was positive, in some in high dilution up to 1/20,000 and in endemic typhus up to 1/4000.

Gear and his colleagues have examined a considerable number of cases of louse-borne typhus (as he says "beyond question one of the major problems confronting the health authorities of South Africa") and his serological findings are summarized as follows:—

1. Agglutinins appear early and, before the end of the first week of illness, the titre has reached diagnostic levels; the maximum titre is reached at the end of the fever and is usually high, 1/12,000 to 1/25,000 are frequently observed.
2. *Proteus OX19* is generally agglutinated to higher titre than *Proteus OX2*. The agglutination of *OXK* is irregular and rarely occurs in high dilutions of serum. Louse borne typhus therefore . . . falls serologically into the same group as classical European typhus.

As regards the effects of the virus on experimental animals it is noted that pneumonic changes in the lungs of guineapigs were frequently observed. Rickettsia were seen in the exudate from the spleen and orchitis although observed was very rare.

Only one case of murine typhus was investigated; this was an accidental infection in the laboratory, the writer himself, and has

already been reported in this *Bulletin*, 1938, Vol. 35, p. 787. The virus was isolated from the blood and passaged in guineapigs. Orchitis was noted in over 90 per cent. of the animals inoculated.

The author then refers to the classical type of tick bite fever as previously described by PIJPER and others. This is a very mild fever with constant primary sore and is carried by larval ticks and is contracted on the open veldt. The author, however, has noted recently in the city and suburbs of Johannesburg another type of the same disease which differs from the above description in several particulars. The fever is severe with delirium and on at least two occasions proved fatal; the primary sore is not constantly present but when found is a typical *tache noire* with lymphadenitis; this sore has been observed to occur in the conjunctiva. The disease is carried by the dog tick from the dog to its owner and in these particulars closely resembles boutonneuse fever of North Africa. Gear is of opinion that these are two types of the same disease, the differences being due to the fact that in one case young people are infected by larval ticks (*A. hebraeum*) from veldt rodents, in the other type older people are infected by dog ticks (*H. leachi*) from domestic dogs.

The author succeeded in cultivating the virus in egg media and noted that in certain cells the Rickettsia had invaded the nuclei of the cells as do the Rickettsia of Rocky Mountain fever. It was also found that the viruses of louse-borne typhus and endemic typhus are immunologically identical as are the viruses of the two types of tick bite fever. Tick bite fever virus does not protect against the typhus viruses but typhus virus shows some degree of protection against tick bite fever virus.

His conclusions are as follows :—

In clinical symptoms, serological reactions and course of the disease in experimental animals South African louse-borne typhus resembles the classical disease and the one case of murine typhus examined resembled cases of murine typhus described in other parts of the world.

The South African variety of tick typhus—tick bite fever—resembled other varieties of tick typhus, namely Rocky Mountain spotted fever, and fièvre boutonneuse, in the serological reactions and in the morphology and distribution of the Rickettsia; especially noteworthy were the nuclear infections seen in tissue and egg cultures. The disease is milder in South Africa than elsewhere. *D. H.*

GEAR (James). **Complications in Tick-Bite Fever. A Survey of Fifty Cases.**—*South African Med. Jl.* 1939. Jan. 14. Vol. 13. No. 1. pp. 35–36.

Tick bite fever as observed by the author in the Witwatersrand is a severe and on occasions a fatal disease, in which the following complications have been met with : general thrombosis, pulmonary embolism, bronchopneumonia, retinal haemorrhages, slight albuminuria in most cases, and in one case with previous history of renal disease, uraemia. Severe conjunctivitis was noted in three cases and was due to primary infection in the eye caused by blood from infected ticks removed from dogs.

Three fatal cases were reported and two other fatal cases were investigated. In one the Weil-Felix reaction was positive, there was a profuse and typical typhus rash and a definite primary sore was

noted on the head with inflamed glands in the neck ; in the second fatal case the blood of the patient infected guineapigs and a virus was isolated. *D. H.*

FITZPATRICK (Florence K.). **Agar-Slant Tissue Cultures of Spotted Fever Rickettsiae.**—*Proc. Soc. Experim. Biol. & Med.* 1938. Dec. Vol. 39. No. 3. pp. 501–502. With 1 chart.

A case of spotted fever Eastern type was reported from Cape Cod, Massachusetts. Blood was taken on the 8th day of the illness and injected into two guineapigs—one of these developed fever.

The virus was passaged to other animals, these reacted with fever and scrotal reaction but no necrosis : there was no mortality among the inoculated animals but later these were shown to be immune to a virulent strain of Rocky Mountain fever. The tissues of infected guineapigs were cultured on agar slopes and subcultured by means of mouse embryo tissue. There was a very rich growth of Rickettsia and guineapigs were infected from the cultures. *D. H.*

BAUERSFELD (E. Herbert). **Rocky Mountain Spotted Fever with Unusual Features.**—*Jl. Amer. Med. Assoc.* 1939. May 6. Vol. 112. No. 18. pp. 1819–1820.

The unusual features in this case were that the rash resembled a typhus rash rather than spotted fever. The rash did not appear till the 9th day instead of the 4th or 5th, it was confined to the trunk and was petechial ; and the patient also developed jaundice, which is not usually seen in the Eastern type of spotted fever. There were no other cases in the neighbourhood but the patient's husband had returned from hitch-hiking in Florida 10 days before the illness commenced and she had removed ticks from his person. The patient died. *D. H.*

PHILIP (Cornelius B.) & DIAS (Emmanuel). **Rocky Mountain Spotted Fever. Failure of Triatomid Bugs to transmit the Virus Experimentally.**—*Mem. Inst. Oswaldo Cruz.* 1938. Vol. 33. No. 4. pp. 473–476. [Portuguese version pp. 469–472.]

The Montana strain of the virus of Rocky Mountain fever was employed in these experiments and bugs of four different genera were tested. Transmission was attempted by feeding other bugs on infected guineapigs and then feeding on clean guineapigs after varying intervals up to 100 days ; interrupted feeding was also tried ; and the fed bugs were also ground up and injected.

The result of the experiment shows that these bugs can neither transmit the virus by their bites nor retain virulent virus in their bodies for longer than 2 to 4 days. [See also this *Bulletin,* 1938, Vol. 35, p. 786.] *D. H.*

DE MAGALHÃES (Octavio). Tifo exantematico em Minas Gerais—O virus. Algumas propriedades. [**Properties of the Virus of Exanthematic Typhus in Minas Geraes.**]—*Acta Med.* Rio de Janeiro. 1939. Apr. Vol. 3. No. 4. pp. 203–216. With 2 charts. English summary.

" On the strength of his own experiments the author concludes that the exanthematous typhus virus of Minas Gerais is of an easy manipulation. It is easy to be isolated from the blood both of human

patients and of inoculated animals, when in the febrile period. When they are in the incubation period, their blood, as a rule, is not infectious, or rarely produces transient and benignant disease. There were exceptional cases in which the blood was infectious 24 hours after inoculation. In human blood the author found the virus up to the 18th day of the disease, on examining the rare surviving cases. The author encountered the virus, in natural state, in wild rabbits, 'preá,' opossum, ticks (A. cajennense) and bedbugs (C. lectularius). He states that the spleen is the organ which contains the largest quantity of the virus. In the central nervous system the virus remains a long time, up to 60 days when the virus is already not found in the circulating blood and the temperature is low. The virus cooled down to –5° or –10°C. has kept its initial virulence up to 160 days after its removal from the animal. When the author operates on the nervous system, great care has to be taken because there appear tissue changes giving origin to substances which are toxic to the animals. At ordinary temperature the virus lost quickly (96 hours) the virulence. The author verified the passing of the virus through placenta in Rhesus the fetus of which presented typical changes of the disease. He also verified that the virus does not pass through healthy skin even when shaved, and therefore direct contagion should be very rare. At temperatures above 50°C. the virus dies quickly, and at 100° instantaneously. A good medium for the preservation of the virus during some days is glycerinated gelatin, taking care to avoid its contamination by bacteria or fungi. The action of radium or X-rays on the virus is none or nearly none. The virus dried in vacuum is able to be virulent up to 7 days, but there are variations in accordance with the sample. The reinoculation of the virus through transmitting arthropods considerably increases its virulence. The virus seldom passes through an L5 filter candle. Employing the current technics, it is cultivated very well on the chorio-allantoid membrane of the chick's embryo."

DE MAGALHÃES (Octavio). Tifo exantematico em Minas Gerais. Diagnostico. (13a comunicação.) [**Typhus Fever in Minas Geraes. Thirteenth Communication. Diagnosis.**]—Reprinted from *Acta Medica*. Rio de Janeiro. 1939. Jan. Vol. 3. No. 1. 13 pp. With 1 chart. English summary (8 lines).

Diagnosis of typhus fever is based on: (1) Clinical signs and symptoms; (2) Epidemiological state; (3) Laboratory tests; (4) Pathological anatomical findings; (5) Isolation of virus and inoculation into a susceptible animal. Considering the question generally the author concludes that in severe and typical cases clinical evidence is enough, but in mild, abortive, or hyperacute cases (*Typhus siderans*) such evidence is not satisfactory and there is need of one or more of the other investigations noted. *H. H. S.*

COOLEY (R. A.). **The Genera *Dermacentor* and *Otocentor* (Ixodidae) in the United States, with Studies in Variation.**—*National Inst. Health Bull. No. 171.* Washington. 1938. Dec. pp. v+89. With 30 plates (9 coloured) & 8 figs. [Bibliography.] 1938. Washington: Govt. Printing Office. [$1·25.]

BRUNEAU & CHAPUIS. Observation et étude expérimentale d'un cas de tsutsugamushi à Hanoi. [**Observation and Experimental Study of a Case of Tsutsugamushi Fever in Hanoi.**]—*Rev. Méd. Française d'Extrême-Orient.* 1938. Aug.–Sept. Vol. 20. No. 7. pp. 907–912. With 7 charts.

A severe case of fever lasting 18 days with a typical primary sore in the umbilical region. Two guineapigs were inoculated with blood

taken on the 14th day of the fever and both died; post-mortem examination showed the usual signs of tsutsugamushi infection. The Weil-Felix reaction of the blood of the patient was positive for *Proteus OXK* 1/1000 but negative for *OX19* and *OX2*. Rabbits inoculated with blood into the anterior chamber of the eye gave the typical reaction of iridocyclitis and keratitis with continued fever. *D. H.*

GUNTHER (Carl E. M.) & SCHROEDER (A. G.). **Further Observations on Endemic Typhus in New Guinea.**—*Med. Jl. Australia.* 1939. May 6. 26th Year. Vol. 1. No. 18. pp. 688–691. With 1 map.

In the appendix to this paper the synonym for endemic typhus is "Japanese River Fever."

Two further cases are described in the present paper, both in white people, in one case which was fatal the Weil-Felix reaction was positive for *Proteus OXK* in a dilution of 1/2500. Since 1934, 46 cases have been reported in this district with a mortality of 20 per cent. All the cases were in white people: none has so far been reported in natives. The whole district is heavily infested with mites and also with bush rats. With a view to prevention, clearing of paths is suggested and removal of mites by bathing: if a primary sore is detected excision of the ulcer is recommended. *D. H.*

WIJERAMA (E. M.). **Notes on Two Cases of Tropical Typhus.**—*Jl. Ceylon Branch Brit. Med. Assoc.* 1938. Nov. Vol. 35. No. 6. pp. 467–468.

Two cases of fever without rash or primary sore; both were dark-skinned people, one an Indian, the other a Sinhalese. The Weil-Felix reaction was positive in both cases in a dilution of 1/5000 for *Proteus OXK*, negative for *OX19*. A guineapig inoculated with the blood of one case developed fever and orchitis and Rickettsia were demonstrated in films from the tunica. *D. H.*

KAWAMURA (Rinya) & YAMAMIYA (Chuzo). **On the Tsutsugamushi Disease in the Pescadores.**—*Kitasato Arch. Experim. Med.* 1939. Jan. Vol. 16. No. 1. pp. 79–91. With 5 figs. on 2 plates.

Thirty-two cases of tsutsugamushi fever have been investigated in the Pescadore Islands. These islands have a low rainfall and little vegetation and conditions are entirely different from the flooded areas where the fever occurs in Japan, and resemble rather the conditions in Sumatra and Malaya.

Notes of 3 cases are given, one mild, one medium and one severe and fatal; a primary sore occurred with lymphadenitis in all cases. The Weil-Felix reaction was positive with *Proteus OXK*.

The authors consider that the disease is identical with that met with in Japan but the mortality rate is much lower. [See this *Bulletin*, 1938, Vol. 35, p. 374.] *D. H.*

LAURENS (J. R.), FORT (P. C. L.) & BERNIER (G. L.). La vaccination contre le typhus exanthématique. L'expérience marocaine (1937–1938). Application en milieu militaire à Casablanca. [**Vaccination against Epidemic Typhus in Morocco.**]—*Rev. Service Santé Milit.* 1939. Feb. Vol. 110. No. 2. pp. 157–195. With 19 charts.

A review of the question from the military point of view. Only a few cases were met with but the authors found that although all

their men or nearly all had been vaccinated, cases of typhus occurred among the vaccinated. Some of these were reactions following the administration of the vaccine (mild attacks of murine typhus) but others were cases of typhus (epidemic) in recently inoculated people. These failures they attribute to two causes: (1) that it is impossible to keep the virulence of the vaccine (murine living typhus virus) constant, and (2) that the vaccine if kept too long loses its efficacy. It is suggested that the difficulties might be overcome by using as vaccine the virus in the dried excreta of fleas. *D. H.*

Gaud. La vaccination contre le typhus exanthématique par la méthode de G. Blanc au cours de l'épidémie de 1938 au Maroc. [**Vaccination against Typhus by the Method of G. Blanc during the Epidemic of 1938 in Morocco.**]—*Bull. Office Internat. d'Hyg. Publique.* 1938. Dec. Vol. 30. No. 12. pp. 2751–2758. With 2 graphs.

The epidemic started in November 1937 and vaccination was commenced in December and approximately 150,000 people were inoculated each month in Casablanca so that by May over one million people had been vaccinated [see *Bull. Inst. d'Hygiène du Maroc* above, p. 988].

Conclusions are that in districts where typhus breaks out and the majority of the people have been vaccinated the epidemic is arrested. In mixed populations where Europeans and natives are living together typhus cases are much more numerous among the natives, but if the native population is vaccinated and the Europeans are not then typhus cases are more numerous among the latter; also in places where the people have already been vaccinated cases are few, whereas in non-vaccinated places there are numerous cases and small epidemics. This vaccine has now been tested in Morocco with satisfactory results. [See this *Bulletin*, 1937, Vol. 34, p. 486.] *D. H.*

Mariani (G.). Vaccinazioni contro il tifo esantematico eseguite nel 1938 sull'altipiano etiopico con il vaccino Weigl. [**Weigl's Vaccine in Prophylaxis of *Typhus exanthematicus* in the Ethiopian Highlands in 1938.**]—*Ann. d'Igiene.* 1939. May. Vol. 49. No. 5. pp. 316–322.

Over 13,000 vaccinations were performed, 2,404 of the subjects being military and 10,672 civilians. Three injections were given at 3-day intervals. In the vast majority the reactions were very slight; locally, in 95 per cent., there was slight redness and swelling with a little pain, coming on 6–12 hours after and lasting 2–4 days, less marked after the second and third injections. As for general reactions 20–25 per cent. showed a rise of temperature of ½–1 degree and some headache; rarely did the fever exceed 38·5°C. A few cases were seen among the vaccinated and these ran a mild course with one exception; in this case death occurred but infection had been acquired before vaccination was begun. Four others were down with the disease before the vaccination was completed, that is, they were vaccinated in the incubation period. *H. H. S.*

ZINSSER (Hans), FITZPATRICK (Florence) & WEI (Hsi). **A Study of Rickettsiae grown on Agar Tissue Cultures.**—*Jl. Experim. Med.* 1939. Feb. 1. Vol. 69. No. 2. pp. 179–190. With 3 charts. [11 refs.]

Agar slopes are prepared with double strength Tyrode solution with horse serum. Small pieces of infected tissue were placed on the surface of the agar and profuse growth of Rickettsia were obtained both of murine typhus and historical typhus. These Rickettsia are readily stained by carbol fuchsin and counterstained with methylene blue [see paper by ZIA, LIU & PANG above, p. 992].

Subcultures were readily obtained by mixing normal mouse embryo tissue with the infected tissues and inoculating new tubes with this.

Inoculations with cultures after 18 months always produced typical reactions in guineapigs and these animals when tested later were immune to passage virus.

Animals inoculated with phenol or formol killed vaccine from agar cultures were also immune to test doses. By this method of culture large amounts of Rickettsia of classical typhus can be obtained and vaccines prepared. [See also this *Bulletin*, 1938, Vol. 35, p. 363.]

D. H.

VEINTEMILLAS (Felix). **Vaccination against Typhus Fever with the Zinsser-Castaneda Vaccine.**—*Jl. Immunology.* 1939. May. Vol. 36. No. 5. pp. 339–348.

The vaccine used in this work was an emulsion of formalinized Rickettsia from irradiated rats (Zinsser's method), the strength of the vaccine was 5,000 million organisms per cc. Doses of vaccine were given at weekly intervals to 12 people. These people were later tested with doses of virus using 25 times the minimum infective dose for a guineapig and estimated as 2,000 minimum infective doses for man ; three of these people showed slight fever only. One dose of the vaccine protected guineapigs against the murine virus but not against the classical virus ; but 3 or 4 doses of vaccine protected against this virus also.

It is proposed to use this vaccine on a large scale in Bolivia where prevention by sanitary methods is exceedingly difficult. *D. H.*

MALARIA.

PRÉCIS OF ABSTRACTS IN THIS SECTION.

RUGE (p. 1003) claims to be able to detect differences in virulence between various strains of benign tertian parasites by means of complete tests of liver function.

RUSSELL and MOHAN (p. 1004) describe the staining of oöcysts in living mosquitoes.

VAN THIEL *et al.* (p. 1004) proved by experiment that *A. maculipennis atroparvus* of the Dutch race is definitely zoophilic towards the pig, but that Italian *labranchiae* and *elutus* are anthropophilic. The keeping of pigs is therefore only useful against *atroparvus* in the Netherlands.

Corradetti (p. 1005) details the Anopheles found in different parts of Abyssinia. From extensive dissections Iyengar (p. 1005) found that *A. philippinensis* in the plains of Bengal and *A. minimus* in the foothills, are the most important vectors. Russell *et al.* (p. 1005) record the simultaneous finding of infected *A. subpictus* and *A. vagus* in Madras. In S. Travancore Mathew (p. 1005) found most infection in *A. fluviatilis* and less in *A. varuna* and *A. culicifacies*.

Pinto (p. 1006) gives an account of the damage done in S. America by the introduction of *A. gambiae* from Africa, and of its breeding habits.

Das Gupta (p. 1006) records abnormal forms of *P. vivax* seen in the blood of an infant 15 hours after birth.

Sarrouy and Portier (p. 1007) report an Arab girl with fatty nephrosis, in the aetiology of which they consider that quartan malaria played an important part. Melchior and Özkan (p. 1007) discuss malaria in relation to surgery.

Sokhey (p. 1008) uses Folin's reagent in a test for plasmoquine in tissues, but it cannot be used with urine.

A note is made of a conference of railway malaria inspectors in India (p. 1008).

A review of the literature on clover in relation to malaria is compiled by Roseveare (p. 1008). Manson (p. 1009) discusses the larvicidal action of various Assamese plants.

Brink and Das Chowdhury (p. 1009) show that 0·75 per cent. of ammonium sulphate prevents mosquito breeding, but as a general measure this would be too costly.

Hewitt (p. 1009) details a technique for demonstrating avian malaria parasites in tissue sections. He (p. 1010) concludes from experiments on birds that merozoites of *P. cathemerium* enter young red cells. When these are few in number multiple infections are common and *vice versa*.

Stauber (p. 1010) found that environmental temperature and period of rest and sleep were effective in modifying the time of segmentation of bird malaria parasites. Huff (p. 1010) shows that the segmentation of malaria parasites in birds is not associated with a rise in body temperature such as occurs in man. The rapid fall in numbers of *P. cathemerium* at the crisis of the initial attack was shown by Boyd (p. 1011) to be due to lowered multiplication and increased destruction which are evident after the third or fourth day.

Manwell and Goldstein (p. 1011) consider that exoerythrocytic schizonts are merozoites accidentally ingested by cells of the reticulo-endothelial system.

Rodhain (p. 1011) found astreptine to be efficacious in *P. knowlesi* infections of monkeys. Chopra and Das Gupta (p. 1012) consider that M & B 693 is more effective than atebrin in *P. knowlesi* infections of apes. *C. W.*

Ruge (H.). Leberfunktion bei frischer Malaria nebst einigen klinischen Bemerkungen. [**Liver Function in Malaria.**]—Reprinted from *Ztschr. f. Klin. Med.* 1939. Vol. 136. No. 3. pp. 311–326.

The author had at his disposal for a complete test of liver function, 47 cases of malaria, including natural and artificial infections with the three varieties of parasite. Estimations of the non-protein nitrogen, serum bilirubin, as well as the application of the laevulose tolerance test, were carried out at the beginning, in the middle and the end of

the attack as well as a few days after its cessation. A comparison of the two strains of benign tertian parasite used showed distinct differences in virulence. The Hamburg strain produced much more pronounced changes than the Madagascar strain. The author concludes from the results of his investigations that with the help of the methods he employed it is possible to estimate differences in virulence of various strains of malarial parasites. Clinical investigation failed to detect any difference in virulence. The atebrin-plasmoquine treatment acted beneficially on the changes occasioned by the malarial infection.

E. D. W. Greig.

Russell (P. F.) & Mohan (B. N.). **Staining Malaria Oocysts in Living Mosquitoes.** [Research Notes.]—*Jl. Parasitology.* 1939. June. Vol. 25. No. 3. pp. 278–279.

In mosquitoes fed for two days on a 10 per cent. solution of glucose with a small quantity of eosin, the oöcysts were clearly stained and readily seen on dissection. Sporozoites were not stained. *C. W.*

van Thiel (P. H.), in collaboration with J. Reuter, J. Sautet & L. Bevere. **On Zoophilism and Anthropophilism of Anopheles Biotypes and Species.**—*Riv. di Malariologia.* Sez. I. 1939. Vol. 18. No. 2. pp. 95–124. With 6 figs. on 2 plates. [28 refs.]

There are real differences in the feeding preferences of the races of *A. maculipennis*; *atroparvus* being a zoophilic race, *labranchiae* and *elutus* anthropophilic. But laboratory experiments on these preferences may give quite misleading results.

By means of experiments with an "artificial arm" exposed in a cage of mosquitoes the authors show that these are attracted by warmth, moisture, carbon dioxide and possibly the odour of blood. But the conclusions to be drawn from these experiments concern only the stimuli inducing attraction and feeding at a distance of some 25 cm. from the host. Experiments were then made in which mosquitoes were placed in a small cage connected by means of sections of stove pipe 1 m. long to two wooden boxes. A pig was placed in one box, a man in the other. Under these conditions the pig attracted many more *atroparvus* than the man. But this result was dependent chiefly on temperature and humidity: if the humidity in the human box was increased with wet towels "even v. Th. could attract . . . . more mosquitoes than a large pig of 100 Kg." An electric heater with wet towels was equally attractive.

The authors conclude, however, that these experiments are too unnatural to give a true index of zoophilism. They therefore provided the two boxes with traps and placed them, one containing a man, the other a pig, in a large muslin cage with free flying mosquitoes. Under these circumstances the number of *atroparvus* of the Dutch race attracted to the pig was twelve times the number attracted to man, and this difference could not be overcome by artificial changes in temperature and humidity. It appears to indicate a true zoophilism. Experiments with the same apparatus in Italy showed that man would attract 2·2 times more *labranchiae* and 2·4 times more *elutus* than the pig. These races appeared definitely anthropophilic. The authors therefore conclude that the provision of pigsties is unlikely to afford protection to man against *elutus* and *labranchiae* in Italy, though

undoubtedly valuable against *atroparvus* in the Netherlands. [This is a fuller account of work already abstracted in this *Bulletin*, 1939, Vol. 36, p. 778.] *V. B. Wigglesworth.*

Corradetti (Augusto). Notizie preliminari sulla fauna anofelica della regione di Gondar, del Lago Tana e della regione del Semien. [**Note on Anophelines of Gondar, Lake Tana and Semien Regions, (Abyssinia).**]—*Riv. di Parassit.* Rome. 1939. July. Vol. 3. No. 2. pp. 153–156. With 2 figs. English summary (6 lines).

"In the regions of Gondar and of Lake Tana the following species of Anopheles have been found up to the present: *A. christyi, A. cinereus, A. demeilloni, A. garnhami, A. mauritianus, A. pretoriensis.*

"In the Semien regions the species known up to the present time are the following: *A. gambiae, A. pretoriensis, A. rhodesiensis* var. *dthalisimilis, A. rufipes.*"

Iyengar (M. O. T.). **A Year's Work on Dissection of Anopheles for Natural Malarial Infection.**—*Jl. Malaria Inst. of India.* 1939. Mar. Vol. 2. No. 1. pp. 105–109.

During twelve months 10,713 Anopheles, from ten localities in Bengal in which malaria is endemic, were sent to a laboratory in Calcutta where they were dissected. The species included, in order of frequence, *annularis, philippinensis, aconitus, minimus, vagus, ramsayi, pallidus, hyrcanus* var. *nigerrimus, culicifacies, varuna, maculatus, subpictus, indiensis, kochi, barbirostris, karwari, tesselatus, sundaicus, jamesi, fluviatilis, jeyporiensis* and *leucosphyrus*. Very few of the last eight species were received. The number found infected was 184, 139 of 1,918 *philippinensis*, 43 of 719 *minimus* and 2 of 408 *pallidus*. *A. philippinensis* is the most important vector in the plains of Bengal and *A. minimus* in the foothills.

Norman White.

Russell (Paul F.), Ramachandra Rao (T.) & Jacob (V. P.). *Anopheles subpictus* **Grassi, 1899, and** *Anopheles vagus* **Dönitz, 1902, found naturally infected with Malaria Plasmodia in South-Eastern India.**—*Jl. Malaria Inst. of India.* 1939. Mar. Vol. 2. No. 1. pp. 95-99.

In Ennore, just north of Madras City, 4,728 *A. subpictus* were dissected: two harboured oöcysts. At Pattukkottai in the Tanjore District of Madras, 3,653 *A. subpictus* and 3,128 *A. vagus* were dissected. Two *A. subpictus* were infected, one with oöcysts and the other with both oöcysts and sporozoites. One *A. vagus* was found with a gland infection. Every care was taken with regard to identification. The simultaneous reporting of naturally infected *A. subpictus* and *A. vagus* is indeed an event. *N. W.*

Mathew (M. I.). **Anopheline Transmitters of Malaria in South Travancore.**—*Jl. Malaria Inst. of India.* 1939. Mar. Vol. 2. No. 1. pp. 101–104.

The results of the dissection of 7,882 anophelines in two areas of South Travancore are recorded, one in the foothills with a river, wells, irrigation channels and rice fields, and an annual rainfall of 80 inches; the other situated 600 feet above sea-level with similar anopheline

breeding places but no large irrigation channels, and an annual rainfall of 59 inches. Since 1935 the latter area has been highly malarious.

The oöcyst and sporozoite rates of 2,602 *A. fluviatilis* dissected were 23·7 and 13·0 per cent.; of 429 *A. varuna* 2·3 and 1·6 per cent., and of 1,131 *A. culicifacies* 0·5 and 0·3 per cent. The total number of dissections reported is 7,882. No species other than the above was found infected. *A. jeyporiensis* was the most numerous, 3,559 dissected. *N. W.*

Pinto (Cesar). Disseminação da malária pela aviação; biologia do *Anopheles gambiae* no Brasil. [**Dissemination of Malaria by Aircraft; Biology of *A. gambiae* in Brazil.**]—*Bol. Biol.* S. Paulo. 1939. May 31. Vol. 4. (N. S.). No. 2. pp. 196–207. With 3 figs. (1 map). English summary.

The introduction of *A. gambiae* into South America was probably effected by shipping, but aircraft may be important in its further spread. The habits of this species are the same as in Africa.

The author considers that it was the introduction of the rapid steamers crossing from Dakar (Africa) to Natal (Brazil) in less than three days which led to the conveyance of *A. gambiae* to South America between August 1928 and February 1930. Its appearance was followed by a local malaria epidemic in which 62 per cent. of wild caught females of this species were infected. In 1938 a widespread epidemic occurred throughout a great part of northern Brazil in which the author gives the number of cases as nearly 40,000 and the deaths as more than 20,000. The mosquito is of the type form of *A. gambiae*. It breeds, as in Africa, in collections of clear stagnant fresh water exposed to sunshine, particularly if aquatic vegetation is present; but also in watering troughs, residual pools in river beds, washing-tubs, etc. It cannot develop in water containing 30 per cent. of sea water. The author stresses the importance of adopting measures to prevent the further dissemination of this insect by means of aircraft. [See this *Bulletin*, 1939, Vol. 36, p. 680.]

V. B. Wigglesworth.

Chédécal (M.). Contribution à l'étude du comportement trophique des anophélinés, à Hanoi. [**Feeding Preferences of Anophelines in Hanoi.**]—*Ann. de Méd. et de Pharm. Colon.* 1939. Jan.–Feb.–Mar. Vol. 37. No. 1. pp. 62–66.

This paper is identical with that published in another journal and abstracted in this *Bulletin*, 1939, Vol. 36, p. 779. *N. W.*

Das Gupta (B. M.). **Some Anomalies in the Morphology of *Plasmodium vivax* occurring in a Newborn Baby.**—*Indian Med. Gaz.* 1939. May. Vol. 74. No. 5. pp. 273–274.

A single blood smear made from an infant fifteen hours after birth on the Bhagatpur Tea Estate (Dooars, India) showed a heavy infection with a malarial parasite which resembled in most respects *Plasmodium vivax*, which had been found in the mother's blood, but differed in that the mature schizonts did not produce more than 12 merozoites. Owing to the fact that infected corpuscles frequently showed fimbriated

margins there was some resemblance to *P. ovale*. The conclusion, however, was reached that the parasite was an abnormal form of *P. vivax*. An excellent coloured plate illustrates the characters of the parasite. *C. M. Wenyon.*

Sarrouy & Portier. Néphrose lipoidique et paludisme. [**Fatty Nephrosis and Malaria.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1939. Apr. 3. 55th Year. 3rd Ser. No. 11. pp. 561–566.

This is a detailed description of the case of an Arab girl, aged 14, admitted to hospital in Algiers with generalized oedema and bad general condition. A year previously violent daily attacks of fever with shivering were followed by painful swelling of the lower extremities. Thereafter attacks of fever occurred at irregular intervals but the swelling persisted. The general condition was not seriously impaired, however, and the child lived a normal life, untreated. A month before admission the condition was aggravated, daily fever occurred with generalized oedema and diarrhoea. The albuminuria varied at different examinations from 4 to 28 grammes per litre. There was abundant urine : from 30 to 35 grammes of urea were passed a day. Blood examination revealed the albumin content down to 40 gm. per 1,000 with inversion of the albumin/globulin ratio, increase of total lipoids to 20·4 gm., and an increase of cholesterol from 1·70, on admission, to 7·50 gm. Neither casts nor blood cells could be found at any time in the centrifuged urine. Blood chlorine remained within normal limits even at times of greatest oedema. Blood urea, generally low among vegetarian natives, was as high as 0·77 gm. per 1,000 on admission. Ambard's constant was normal and arterial tension was not high. In the blood were found rosettes of *P. malariae*, but no young forms. Malarial treatment followed by an intensive treatment with raw calf's liver effected some improvement. Blood urea fell, the oedema diminished and there was an improvement of the cardiac condition. The patient left hospital before thyroid treatment, which was to have been tried, could be given.

The author considers that malaria played an important part in the aetiology of this fatty nephrosis syndrome in which the renal function was unimpaired. *N. W.*

Melchior (Eduard) & Özkan (Adil). Malaria und Chirurgie. [**Malaria and Surgery.**]—*Schweiz. Med. Woch.* 1939. July 15. Vol. 69. No. 28. pp. 640–642.

The authors are working in Turkey and in Anatolia, from which district their clinic receives cases ; malaria plays a very important rôle. They discuss the subject under four headings. Firstly, they consider the provocation of malaria by operations and accidental trauma : the differential diagnosis is particularly important when the malarial attack occurs in persons with infected wounds. As the malarial parasites may not be found in the peripheral blood in all cases it may be necessary to fall back, in such cases, on a therapeutic diagnosis by administration of quinine. Secondly, the confusion of acute appendicitis with an attack of malaria is dealt with and a case illustrating this point is detailed. Thirdly, the surgery of the malarial spleen is discussed : the authors consider that the chief indications for splenectomy are, torsion and splenic rupture. They do not favour splenectomy in malarial cachexia. Lastly, the influence of malarial

cachexia on surgical mortality is dealt with. They find that resection of the large intestine is liable to be followed by death in patients suffering from malarial cachexia, and there was no other discoverable cause of death. They note that tabes and general paralysis are rare, probably causally related to the high incidence of malaria in the districts. On the question of whether the rarity of post-operative thrombosis and embolism in their cases can be ascribed to the same cause they are not prepared to express an opinion.

E. D. W. Greig.

Bombay : Report of the Haffkine Institute for the Year 1937 [Sokhey (S. S.), Director].—**A Colorimetric Test for Plasmoquine** [Nandi & Dikshit] p. 44.

If a few drops of Folin's phenol reagent be added to an aqueous acid solution of plasmoquine and the mixture made strongly alkaline with Na_2CO_3, a blue colour develops increasing to maximum intensity in about half an hour. The test can detect plasmoquine in a dilution of 1 in a million and can be used for quantitative estimations in dilutions up to 1 in 200,000. The test can also be used for the detection and estimation of plasmoquine in tissues if the latter be treated with an excess of NaOH and extracted with ether to get rid of phenolic compounds, the sodium salts of which are very soluble in water but insoluble in ether. The test cannot be used for the detection of plasmoquine in the urine. [See also this *Bulletin*, 1939, Vol. 36, p. 580.] *N. W.*

Khargpur, B.-N. Railway : **Report of the Proceedings of the Third Annual Conference of the B.-N. Railway Malaria Inspectors held on the 21st February, 1938.**—88 pp. 1938. Calcutta.

Like most reports of conferences this does not lend itself to summary, nor is a summary generally necessary. Nearly all important additions to knowledge reported to conferences find their way independently into the scientific press. This report of a conference of Malaria Inspectors of the Bengal Nagpur Railway has an interest all its own. Quite apart from the local interest and importance of the observations recorded, which in many instances are very considerable, it reveals a scientific outlook, powers of observation and an enthusiasm possessed by a team of Malaria Inspectors which must rob the arduous daily round of much of the monotony which it would otherwise possess. The subjects discussed include man-made malaria, the yellow fever menace to India, financial aspects of species control, malaria control on railway constructions, resting habits of adult mosquitoes, and anopheline breeding of many of the species of importance in the territory served by the Railway. The discussions on all these subjects were mostly confined to descriptions of personal observations made by contributors, a precedent worth following. *N. W.*

Roseveare (G. M.). **Clover and Malaria.**—Reprinted from *Herbage Reviews*. Aberystwyth. 1939. June. Vol. 7. No. 2. pp. 90–93. [19 refs.]

This is a short review of the subject of Melilotus and its possible action in prevention of malaria. The idea was strongly advocated by

D'Herelle some 12 years ago, but Bruce Mayne's experiments showing that coumarin has no plasmocidal value in mosquitoes and that the low prevalence of malaria in clover districts might be equally well explained by the drainage necessary for proper growth of the clover have now exploded the idea. This review, though bringing forward nothing new, is useful for reference on a subject now almost a matter of history. *H. H. S.*

Manson (D.). **The Action of Certain Assamese Plants as Larvicides.**—*Jl. Malaria Inst. of India.* 1939. Mar. Vol. 2. No. 1. pp. 85–93.

The conclusions of this paper are as follows :—

" Duranta, Zanthoxylum, Gardenia, and Tephrosia are valuable for their larvicidal action and can be used with success in water where there is no high velocity of flow, and in ponds and swamps.

" These plants all grow in Assam in profusion, and can be used as auxiliary larvicides to oil and paris green in suitable situations. Duranta is also an excellent shade plant for *malariogenic drains*. Polygonum is useful as a shade plant for small surface drains. Tephrosia may also be included in the same category. Gardenia and Zanthoxylum, while excellent larvicides, are not useful for shade planting, but only for their larvicidal action. The larvicidal action of acetone dilutions of the seeds of *Tephrosia vogelii* is very marked and is due to Degueline, an isomer of Rotenone."

Brink (C. J. H.) & Das Chowdhury (D. K.). **Ammonium Sulphate as a Combined Fertilizer and Mosquito Larvicide.** [Abstract.]—*Jl. Malaria Inst. of India.* 1939. Mar. Vol. 2. No. 1. pp. 111–112.

Experiments have shown that mosquito breeding cannot take place in water that contains 0·75 per cent. or more of ammonium sulphate. Observations were made with *Culex fatigans* and *A. stephensi*. Questions of cost would prohibit attempts to control mosquito breeding with ammonium sulphate in any large irrigation area. *N. W.*

Hewitt (Redginal). **A Staining Technic for Demonstrating Avian Malaria Parasites in Tissue Sections.**—*Amer. Jl. Hyg.* 1939. May. Vol. 29. No. 3. Sect. C. pp. 115–117.

The technique described consists of fixation in Zenker's fixative containing 5 per cent. of formol, the mordanting of the sections in 2·5 per cent. potassium bichromate solution for one-half to one hour and, following a quick wash in distilled water, staining for 24 hours with Wolbach's Giemsa mixture (distilled water 100 cc., 0·5 per cent. Na_2HCO_3 [so given in the text, ? $NaHCO_3$] solution 2–4 drops, methyl alcohol 3 cc., Giemsa stain 2·5 cc.). The sections are washed in distilled water coloured lemon yellow with the above bichromate solution, differentiated in 70 per cent. alcohol (30 seconds to 2 minutes) and then dehydrated with graded xylol acetone mixtures up to pure xylol and mounted in balsam or cedar oil. *C. M. Wenyon.*

Hewitt (Redginal). **Experimental Erythroblastosis in Canaries and its Effect on Infections with** *Plasmodium cathemerium.*—*Amer. Jl. Hyg.* 1939. May. Vol. 29. No. 3. Sect. C. pp. 135–148. With 2 figs. [15 refs.]

By the administration of phenylhydrazine solution to canaries, either orally on a number of days or intravenously on one day, a rapid destruction of red blood corpuscles is brought about, followed by the appearance in the blood of large numbers of immature red cells. Birds with this experimental erythroblastosis were inoculated with *Plasmodium cathemerium* and the resulting infection was compared with that produced in normal canaries. It was noted that in normal birds few young red cells are present, while the number of cells with multiple infections is great at the start. The cells with three or more organisms are destroyed during their growth, only those cells with one or two organisms surviving. In the phenylhydrazine treated birds there are many young red cells and in consequence only few with multiple infections. There is thus less parasite destruction and a more rapid rise in the parasite number which reached a higher peak than in the normal birds. The argument is that the merozoites enter young red cells. When these are few in number multiple infections are common, whereas when they are plentiful multiple infections are rare. [See also this *Bulletin*, 1939, Vol. 36, p. 585.]

C. M. W.

Stauber (Leslie A.). **Factors influencing the Asexual Periodicity of Avian Malarias.**—*Jl. Parasitology.* 1939. Apr. Vol. 25. No. 2. pp. 95–116. With 7 figs. [31 refs.]

Working with strains of bird malaria parasites which showed some regularity in the synchronism of reproduction, attempts were made to modify the time of segmentation by changes in temperature, light, feeding, host activity and rest and it was found that the environmental temperature and the period of rest and sleep were effective. Light acted only through the eye in that it rendered the host active. The young trophozoites were the forms of parasite chiefly influenced and it was the effect of the changes on them which determined the change in the time of subsequent schizogony. As there is a marked difference in the temperature of the active and the resting canary it seems that this temperature change, which occurs when the bird passes from a condition of rest to one of activity or *vice versa*, or some physiological condition which underlies it is the factor which determines the actual synchronism. *C. M. W.*

Huff (Clay G.). **Relations between Malarial Infections and Body Temperatures in Canaries.**—*Amer. Jl. Hyg.* 1939. May. Vol. 29. No. 3. Sect. C. pp. 149–154. With 1 fig. [11 refs.]

Though considerable fluctuations in the temperature of birds occur normally, a notable feature being a rapid rise of temperature on awakening, it was not possible to show that the presence of reproducing malarial parasites influenced the temperature. Four strains of parasite were tested in canaries but there was a complete absence of paroxysmal temperature rises at the time of segmentation of the

parasites. This is in striking contrast to what obtains in man and mammals generally, where the segmentation of malarial parasites is associated with marked rises in temperature. *C. M. W.*

Boyd (George H.). **A Study of the Rate of Reproduction in the Avian Malaria Parasite,** *Plasmodium cathemerium.*—*Amer. Jl. Hyg.* 1939. May. Vol. 29. No. 3. Sect. C. pp. 119 129. With 3 figs. [11 refs.]

Working with *Plasmodium cathemerium* infections in canaries the author has observed that the largest groups of merozoites are formed by schizonts during the early stages of an infection. The early schizonts produce on an average about 16 merozoites, but the size of the schizonts diminishes till by the fourth day only about 12 merozoites are produced. There is a slight increase on the seventh day, but the original number of 16 merozoites is not again reached. Parasite destruction is low at the commencement of an infection but on the third day may involve 90 per cent. of the parasites produced. This level of destruction was maintained throughout the period of observation. Thus the rapid fall in numbers of parasites at the crisis of the initial attack is due to the combined effect of lowered multiplication rate and increased destruction. *C. M. W.*

Manwell (Reginald D.) & Goldstein (Frederick). **Exoerythrocytic Stages in the Asexual Cycle of** *Plasmodium circumflexum.*—*Amer. Jl. Trop. Med.* 1939. May. Vol. 19. No. 3. pp. 279–295. With 3 plates. [17 refs.]

The paper describes the presence of exoerythrocytic schizonts in the case of *P. circumflexum* infections in canaries. This brings to five the number of species of bird malarial parasite in which these forms have been found, the others being *P. cathemerium, P. praecox* (*relictum*), *P. elongatum* and *P. gallinaceum,* all except the last named being inoculable to canaries. The authors express the opinion that the occurrence of these schizonts is accidental and is due to the chance ingestion of merozoites by the cells of the reticulo-endothelial system. The same merozoites if they had entered red blood corpuscles would have developed into ordinary pigmented schizonts. It is thus the host cell which determines the size of the schizont and the number of merozoites produced. *C. M. W.*

Rodhain (J.). Paraaminophenylsulfamide et plasmodium des singes. [*p*-**Aminobenzinesulphonamide and Monkey Plasmodium.**]—*Ann. Soc. Belge de Méd. Trop.* 1938. June 30. Vol. 18. No. 2. pp. 255–258.

During the treatment of two chimpanzees for a bronchitic infection with astreptine (*p*-aminobenzinesulphonamide) it was observed that a simultaneous infection with malarial parasites of the *P. falciparum* type (*P. reichenowi*) was eradicated. This chance observation led the author to test the action of the drug on *P. knowlesi* infections in rhesus monkeys. It was found to be equally efficacious in its action against this parasite, though not so active as atebrin. *C. M. W.*

Chopra (R. N.) & Das Gupta (B. M.). **'M. & B. 693' (2-Sulphanilylaminopyridine) in Ape Malaria.**—*Indian Med. Gaz.* 1939. Apr. Vol. 74. No. 4. pp. 201–202.

The conclusion reached by the authors is that M. & B. 693, unlike prontosil and soluseptasine, is capable of curing monkeys of a *Plasmodium knowlesi* infection, as judged by the failure to relapse (examination of thick films) after a five-day course. The drug was administered intramuscularly in oily suspension, the daily dose varying from 0·05 gm. to 0·15 gm. It appears to be superior to atebrin for this infection for, though there is a marked immediate action with atebrin, relapse almost invariably occurs in 10 to 15 days with rapid multiplication of parasites and death of the host if further treatment is withheld.

C. M. W.

LEPROSY.

Précis of Abstracts in this Section.

Lowe (p. 1013) gives an account of certain leprosy legislation introduced in India at the end of the 19th century. Pooth (p. 1014) gives an account of leprosaria in mediaeval West Pomerania.

Lowe and Chatterji (p. 1015) studying seasonal variations in clinical and bacteriological activity found this to be increased in the hot season in Calcutta, to end abruptly with the onset of the rains.

In a region of the Congo Degotte (p. 1016) found a disease rate of 44·37 per 1,000. This high rate is favoured by deficient sanitation and prolonged contact with cutaneous cases. In Panama, beyond the Canal Zone, the rate found by Courtney (p. 1016) was 0·26 per 1,000. The disease appears to be familial and he postulates inherited predisposition in addition to prolonged contact.

In New Caledonia Kervingant and Baré (p. 1016) conclude from a recent survey that leprosy is stationary in the natives and diminishing in the Europeans. The improved methods of prophylaxis include 10 agricultural colonies with good social and medical facilities. The outlook is much better than formerly. Cilento (p. 1017) shows that in Queensland more cases have been detected recently than before owing to legislation giving power to examine suspects and contacts. There was a serious outbreak in Western Australia in 1933–1936 but Davis (p. 1017) reports that it is now well under control.

Innes (p. 1018) reports a hopeful attempt to control leprosy in the British Solomon Islands by means of a colony system, with outlying dispensaries, as at Uzuakoli in Nigeria.

Muir (p. 1018) considers that leprosy attacks especially primitive people coming into social and industrial contact with more civilized races.

Ryrie (p. 1018) advises a mobile leprosy unit for surveys in Malaya, and points out that compulsory institutionalism is responsible for much concealment.

Bertrand *et al.* (p. 1019) working with Kedrowsky's bacillus conclude that it is the bovine type of *Myco. tuberculosis.*

Tisseuil (p. 1019) succeeded in grafting skin infected with tuberculoid leprosy into other infected patients, giving rise to new tuberculoid

lesions. He failed to infect rats with human leprosy material emulsified in a number of different ways. Suzuki (p. 1019) reports increase in numbers of leprosy bacilli after intraperitoneal inoculation of animals.

McKinley (p. 1020) reviewing the history of attempts at cultivation of the leprosy bacillus, considers that up to 1917 the organisms grown could not be regarded as the true leprosy organism. He describes his own technique and Scott in comment carries the account further to include the Calcutta experiments and McKinley's own recent work. Suwo and Kin (p. 1021) report multiplication of leprosy bacilli in tissue cultures of leprosy nodules. Rousseau and Gaugeat (p. 1021) claim to have cultivated the human leprosy bacillus on Loewenstein's medium. Evans (p. 1021) isolated chromogenic mycobacteria, on Clegg's medium, which are considered possibly to be leprosy bacilli.

Oberdörffer (p. 1021) repeats his theory that the plant *Collocasia antiquorum* predisposes to leprosy when used as food. Lowe and Chatterji (p. 1015) were unable to confirm this hypothesis in Calcutta.

Radna (p. 1021) discovered that patients with lepromata may excrete leprosy bacilli in the urine if undergoing treatment (for yaws or syphilis) with novarsenobenzol. He (p. 1022) reviews the possible ways by which the bacilli may be eliminated from the body, and their importance in transmission. He further (p. 1022) as a result of house enquiries made regarding lepers, concludes that family contact constitutes the greatest danger and may take place in a number of ways. Hereditary transmission is probably not important [see Courtney above]. *C. W.*

Lowe (J.). **A Curious Chapter of the History of Leprosy in India, the Indian Leprosy Commission of 1890.**—*Leprosy in India.* 1939. July. Vol. 11. No. 3. pp. 82–86.

In 1887 the Government of India decided that efforts should be made to control leprosy and set about obtaining information as to the number of leprosy institutions there were, the numbers of inmates, the rules as regards separation of sexes, etc. Institutions were few and were mostly temporary halting-places for vagrant leper beggars. In order to stamp out the disease the conclusion was reached in 1888 that the sexes would have to be separated and all lepers confined for life. This being, of course, out of the question, the only practicable measure was to give medical and charitable relief in voluntary hospitals and asylums. The following year, however, the Legislative Department prepared a draft bill for compulsory detention of all vagrant lepers, and correspondence on this resulted in the appointing of a Commission to investigate the problem of leprosy in India. This Commission consisted of five members, three selected in England by the National Leprosy Fund authorities [this fund was a memorial to Father Damien who had recently died in Hawaii] and the Royal Colleges of Physicians and Surgeons, and two by the Government of India. The former three were Dr. Beavan N. Rake of Trinidad, Dr. G. A. Buckmaster and Dr. A. A. Kanthack, and the two from India were Surgeon-Majors A. Barclay and S. J. Thompson, I.M.S. Of the first three only Beavan Rake had had any experience in leprosy and he was a man with pronounced anti-contagionist views who first cleared the board by ruling out of consideration all cases in an endemic area, on the grounds that such had no evidential value.

The findings of the Committee were : (1) That leprosy is neither syphilis nor tuberculosis, but has analogies with the latter : (2) That leprosy is not transmitted hereditarily, and the disease tends to die out because the majority of lepers are sterile ; (3) That it is contagious and inoculable but is not spread widely in these ways ; (4) That leprosy is not peculiar to any race or caste, nor does it arise from the use of any particular food, nor by reason of any climatic or telluric conditions ; (5) That predisposing causes are poverty, bad food, and insanitation ; and (6) That it arises *de novo* under a combination of circumstances and conditions whose interrelation is not known.

Consequently, the Commission did not recommend segregation, either absolute or partial, but advocated voluntary isolation. Their recommendations included : (1) Prohibition of lepers as sellers of food or drink, or their engaging in such occupations as barbers and washermen ; (2) Discouragement of concentration of lepers, forbidding vagrant lepers to beg, to use public conveyances, or to frequent places of public resort ; (3) Lepers infringing these regulations were to enter asylums to be built near towns.

Thus we have the peculiar state of things that the disease is contagious to an " exceedingly small " degree, but voluntary isolation, prohibition of certain trades and occupations, control of movements and establishment of asylums are recommended for dealing with it. The Government of India adopted the report and on it was based the Leper Act of 1897 which was clearly framed with the view of deferring to public opinion [that leprosy is contagious] and mitigating a public nuisance. It was a permissive Act, enforceable by local governments by notification. *H. H. S.*

Pooth (Peter). **Leprosaria in Mediaeval West Pomerania.**—*Internat. Jl. Leprosy.* Manila. 1939. Apr.–June. Vol. 7. No. 2. pp. 257–268. With 1 fig.

Mediaeval West Pomerania extended from what is now known as the River Ryck near Greifswald to the border of Mecklenburg and included the island of Ruegen. The author speaks of the 13th century onwards and the existence of leprosaria in the towns and countryside of the mainland and on Ruegen. In the earlier days the lepers of a town, excluded from civil and social life, settled outside the city walls and depended for subsistence on charity. Generally they did so well that others joined them till they formed a community and the town council had to provide for their orderly administration. New buildings were erected, usually with a chapel attached and admission depended on payment of a fee, but there were funds to pay for the poor. Dr. Oberdoerffer who translated the article from the German, studied in Greifswald and knows the district well. He conjectures that the population in the 13th–15th century was not more than 80,000 and nineteen leprosaria are noted and he thinks the incidence was about 5 per mille. In northern Germany, states the author, it was the custom to dedicate these chapels and leprosaria to St. Juergen (St. George), and wherever he finds a St. Juergen church or village he regards that as evidence of the former existence of a leprosarium. [From records which he has traced of the " leprosaria " becoming transformed into monasteries or almshouses in so short a time as fifty years, we can only infer that, as elsewhere, these houses were not reserved only for lepers but were probably inns for travellers

also, and that, by inference, the number of cases in the buildings was not so high as 5 per mille (or 400 interned). The latter figure, if correct, would lead us to expect at least twice that number in all, a 10 per mille incidence clearing up in less than a century. We may call to mind in this connexion Sir W. P. MacArthur's words in his Notes on Old-time Leprosy in England and Ireland. "The mere label 'leper hospital' attached to an institution is no proof that it ever contained a single leper." The Erse word for oratories, he states, was translated into Latin as "nosocomia," that is, "hospitals" and on this being rendered into English the gloss "leper houses" was added so that leper houses became as numerous as oratories.] *H. H. S.*

British Empire Leprosy Relief Association. **Leprosy, a Key Disease. Report for 1938.**—36 pp. With 9 figs. & 2 maps. London: 115, Baker Street, W. 1.

This report reveals steady progress. It deals with the medical secretary's tour in East Africa and gives summaries of the work in other areas. The Association now employs 18 Toc H lay workers in addition to a few medical men and nurses. A noteworthy feature is the receipt of £8,920 18*s.* 9*d.* from the first wireless appeal. The leprosy exhibition has continued to arouse fresh interest and to obtain increased support wherever it has gone. *L. Rogers.*

Lowe (John). **Lecture Notes on Leprosy.**—56 pp. With 14 plates & 2 maps. 1938. New Delhi: British Empire Leprosy Relief Association, Indian Council, Talkotara Road.

This booklet consists of lecture notes used by the author in India for addresses. It should be useful to lay workers and beginners.

L. R.

Lowe (John) & Chatterji (S. N.). **Seasonal Variations in Leprosy in Calcutta.**—*Internat. Jl. Leprosy.* Manila. 1939. Apr.–June. Vol. 7. No. 2. pp. 137–148. With 5 figs.

A visit of Dr. Oberdörffer to Calcutta led these authors to study the seasonal activity of their cases as shown by clinical signs and bacteriological findings. An analysis of 2,779 cases seen within three years showed the highest numbers between March and October affecting the neuromacular cases, but not the lepromatous ones. Clinical activity was greatest in March to September, and the proportion showing bacilli rose markedly to a maximum of 23 per cent. in April and May, but fell suddenly in June to below 5 per cent., with a yearly average of below 10 per cent. This indicates an increase of the bacilli early in the year, followed by increased cellular activity, with a sudden June fall in the bacilli, and a later one in cellular activity. Neither Stein's explanation of sudden meteorological changes nor Oberdörffer's coco-yam hypothesis [see below p. 1021] explains these facts, which appear to be related to increased bacterial activity in the hot season, ending abruptly with the onset of the rains in Calcutta in June. *L. R.*

Degotte (J.). Contribution à l'étude épidémiologique de la lèpre dans la région du Nepoko (Kibali-Ituri). [**Epidemiology in the Nepoko Region.**]—*Ann. Soc. Belge de Méd. Trop.* 1939. Mar. 31. Vol. 19. No. 1. pp. 1–12. With 1 map.

This is a study of leprosy in a badly infected area of the Congo, with heavy rainfall. An examination of 8,294 of the native population revealed 368 definite and 162 suspected leprosy cases, 44·37 and 19·53 per mille respectively. This very high rate was favoured by deficient sanitation and infections through prolonged contact especially with cutaneous cases. Males were most affected and a large proportion of the cases were neural. *L. R.*

Courtney (Kenneth O.). **Leprosy in Panama. A Study of its Origin and Spread.**—*Internat. Jl. Leprosy.* Manila. 1939. Jan.–Mar. Vol. 7. No. 1. pp. 29–40. With 10 figs.

This paper records the results of an inquiry into the occurrence of leprosy in the Panama Republic beyond the limits of the Canal Zone. The infection by the early Spanish settlers is referred to, and details of the present distribution with family trees illustrating infections are then given for each province. Four main towns supplied 40 per cent. of the 109 cases in the Palo Seco Colony and records of the deaths of 120 other cases were found. In addition, 7 active and 13 unconfirmed cases were met with, and the active cases amount to 0·26 per mille of the population. A history of contact infection was traced in 74 per cent. of the cases in the colony, and among new provincial cases, and those visited in hospitals and in their families totalling approximately 116, in all except one prior contact with leprosy was established. The disease appears to be a familial one, and the author regards it as due to an inherited predisposition, coupled with prolonged intimate contact with the disease. *L. R.*

Kervingant (M.) & Baré (J.). La lèpre en Nouvelle-Calédonie en 1936. [**Leprosy in New Caledonia in 1936.**]—*Internat. Jl. Leprosy.* Manila. 1939. Apr.–June. Vol. 7. No. 2. pp. 175–200. With 14 figs.

After references to previous inquiries into the leprosy position in New Caledonia in 1914 and 1924, the authors report on the present more hopeful outlook due to the adoption of modern methods of prophylaxis. In the course of a detailed description of the present incidence of the disease they point out that among the natives the sexes are nearly equally affected and the nerve cases greatly exceed the cutaneous. European male cases number twice as many as females, but this is only in proportion to the numbers of each in the islands. The recognized native cases in 1936 numbered 953, or 31·9 per mille, and the additional suspected cases were 316, or 10·5 per mille. The European confirmed cases numbered 128, 5·2 per mille, and the suspected ones 15 or 0·5 per mille. The authors come to the conclusion that the indigenous cases are stationary in number but the European ones are diminishing.

Of greater interest is the information given regarding the adoption of improved methods of prophylaxis. In 1924 there was a nominal and ineffective isolation of patients in 64 segregation villages with no proper supervision and no treatment. In 1936 there were 10 leper

agricultural colonies with good hygiene, social amenities and a dispensary to supply regular treatment for the contagious cases. The closed uninfective patients are free, are treated in their own villages and are examined every three months. A good colony type of sanatorium has also been supplied for the European patients. The outlook is thus much more hopeful. *L. R.*

Baré (J.). Le service de la lutte contre la lèpre en Nouvelle-Calédonie. [**Leprosy Prophylaxis in New Caledonia.**]—*Ann. de Méd. et de Pharm. Colon.* 1939. Jan.–Feb.–Mar. Vol. 37. No. 1. pp. 165–200. With 12 figs.

This article is on similar lines to that above by the same author with Kervingant. *L. R.*

Cilento (Raphael). **Leprosy in Queensland.**—*Internat. Jl. Leprosy.* Manila. 1939. Apr.–June. Vol. 7. No. 2. pp. 201–208. With 2 figs. (1 map).

This brief paper records the present position of the disease in Queensland. A Health Act of 1937 gave new powers to examine suspects and contacts. An inquiry in a northern area revealed a focus of leprosy of 14 clear and 25 suspected cases, and every infected family is now being examined every three months for new cases. Between 1925 and 1938 infections have been found in 58 whites and 71 aboriginals, and 126, of whom 88 were males and 38 females, have been admitted to the Pearl Island Lazaret. The number of new detections in the five years to 1935 was more than double those found in any of the six previous five-yearly periods, owing to the increased number detected in the coloured people. The more active measures should in time prove of benefit. *L. R.*

Davis (A. P.). **Leprosy in Western Australia.**—*Internat. Jl. Leprosy.* Manila. 1939. Apr.–June. Vol. 7. No. 2. pp. 209–215. With 1 fig.

Leprosy is only found in the northern, warmer part of the area, where an enquiry into its incidence has been carried out during the last two years by Dr. Davis. A spot map shows most cases in the northern hot, humid Kimberley division, and a smaller number in the North-West division. In the former 42 cases were found among 4,001 examined, and in the latter 2 among 1,690 persons. The disease is said to have been introduced by indentured labour of the pearling industry. The total number of notifications in the West Kimberley division rose from 2–7 in the years 1908 to 1932 to 41–58 in 1933 to 1936. The serious outbreak thus revealed is said to be now well under control and the new notifications fell to 19 in 1937. *L. R.*

Vogel (E.) & Riou (M.). Les maladies épidémiques, endémiques et sociales dans les colonies françaises pendant l'année 1937. Lèpre. [**Leprosy in the French Colonies during 1937.**]—*Ann. de Méd. et de Pharm. Colon.* 1939. Apr. Vol. 37. Supplement. pp. 534–543.

This is a further paper giving the number of known leprosy cases in French colonies all over the world, but contains nothing new. In the absence of reliable surveys the numbers are doubtless far below the truth. *L. R.*

Innes (James Ross). **Report of Leprosy Survey of the British Solomon Islands Protectorate.**—70 pp. With 9 maps (3 folding), 1 plan & 1 diagram. 1938. Suva, Fiji.

This is the full report of the inquiry, a summary of which was dealt with in this *Bulletin*, 1938, Vol. 35, p. 882. It brings out the laborious nature of the survey and takes a fairly hopeful view of an attempt to control the disease by the colony system, with outlying dispensaries, as in use at Uzuakoli in Nigeria, while not minimizing the difficulties owing to the bush nature of the most heavily infected island of Malaita.

L. R.

Muir (Ernest). **Leprosy in Africa.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1939. June 29. Vol. 33. No. 1. pp. 119–126.

This address is of a general nature. The author once more considers leprosy to be a disease that especially attacks primitive people on coming into social and industrial communication with more civilized races, including changes of diet, clothing and manner of life. A table is given of the estimated number of leprosy cases in British African possessions, which total 256,000 and is considered to be below the truth. The method of prophylaxis in use in South Africa is a modified form of compulsion, and in Nigeria is a free colony system with outlying treatment dispensaries with the sympathetic help of missionaries and Toc H workers. *L. R.*

Hasselmann (C. M.). Zur Epidemiologie und Sozialhygiene der Lepra, mit besonderer Berücksichtigung der Verhältnisse auf den Philippinen. [**The Epidemiology of Leprosy as seen in the Philippines.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1939. May 10. Vol. 121. No. 5. pp. 649–662. [18 refs.]

This is a general account of the campaign against leprosy in the Philippines on the lines of many previously extracted papers on the subject ; it contains nothing new. *L. R.*

Lafferre & Rouanet. Lèpre autochtone bretonne. Maladie de Morvan ou syringomyélie ? (A propos de deux observations.) [**Indigenous Leprosy in Brittany.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1939. June 26. 55th Year. 3rd Ser. No. 21. pp. 968–973.

Ryrie (G. A.). **Some Principles for directing Leprosy Surveys.**—*Jl. Malaya Branch Brit. Med. Assoc.* 1939. Mar. Vol. 2. No. 4. pp. 233–238.

This brief note is largely concerned with the local conditions necessitating patience and tact in Malaya during leprosy surveys. A card is described for recording the observations. The author emphasizes that a great deal can be done by treatment, especially in tuberculoid leprosy. Attention should be paid to diet in relation to incidence. A leprosy mobile unit is advised for survey propaganda and treatment similar to that long used in India. A large factor in concealment of cases is the " bugbear of compulsory institutionalism." A full list of all known patients should form the basis of the survey.

L. R.

Bertrand (Ivan), Bablet (Jean) & Block (Françoise). Sur l'inoculation intracérébrale au lapin de bacilles acido-résistants isolés chez les lépreux. [**Intracellular Inoculation of Bacilli from Lepers.**]—*C. R. Soc. Biol.* 1939. Vol. 130. No. 14. pp. 1565–1566.

This is a report on the results of intracerebral inoculation in rabbits with acid-fast bacilli obtained from leprosy patients. The strain isolated by Chaussinand at Saigon, that of Levy, of Hyggo and Uchida in Tokyo and of Kedrowsky have been used by the technique the authors had previously employed in the case of tubercle and paratubercle bacilli. The first three forms gave negative results, but Kedrowsky's bacillus produced similar lesions to those caused by paratubercle bacilli, so the authors conclude that this organism is a bovine tubercle bacillus isolated from a leprosy patient infected by both it and the true lepra bacillus. *L. R.*

i. Tisseuil (J.). Essai d'inoculation de la lèpre humaine aux lépreux, par greffe hétéroplastique dermo-épidermique. [**Inoculations of Human Leprosy Bacilli into Animals.**]—*Bull. Soc. Path. Exot.* 1939. Apr. 4. Vol. 32. No. 4. pp. 382–385.

ii. ——. Essais d'inoculation de la lèpre humaine au rat d'élevage. (Première note.)—*Ibid.* pp. 542–545.

iii. ——. Essais d'inoculation de la lèpre humaine au rat d'élevage. (Deuxième note.)—*Ibid.* pp. 546-553.

These papers report further attempts by the author to infect animals with human lepra bacilli. i. This is a record of further grafting of tuberculoid leprosy infected skin into other infected subjects, who are more likely to give positive results than apparently healthy subjects. In two instances at the end of four to six months new tuberculoid leprosy lesions were found. In a third lepra bacilli were still found in the lesions at the edge of the ulcer.

ii. This note records attempts to inoculate human leprosy into bred rats. Emulsions of leprous material were used, with the addition of biological or chemical substances or other acid-fast bacilli. With emulsions the local reaction quickly disappeared. With the addition of other substances reactions of variable strength resulted, and in two a second injection after 6 to 8 weeks produced a black slough. The glands were frequently positive for acid-fast organisms after four months. No results were obtained by ingestion of the leprous material, so the results were all negative as far as reproducing human leprous lesions is concerned.

iii. In this series of experiments the emulsions of leprous material were made up in broth, beef bile, gelatine, olive oil, glycerine and vaseline respectively as culture media favourable to bacillary growth, and other acid-fast bacilli were added in some. No definite results were obtained. *L. R.*

Suzuki (Rissyun). Ueber die Impfung von Tieren mit Leprabazillen. [**Inoculation of Animals with Lepra Bacilli.**]—*Tohoku Jl. Experim. Med.* 1939. July 30. Vol. 36. Nos. 2 & 3. pp. 146–152. With 3 figs. (2 coloured).

This short paper records that intraperitoneal injection of lepra bacilli resulted in the formation of nodules containing the organisms, which are believed to have increased in numbers. *L. R.*

McKinley (Earl B.). **The Bacteriology of Leprosy. A Review.**—*Internat. Jl. Leprosy*. Manila. 1939. Jan.–Mar. & Apr.–June. Vol. 7. Nos. 1 & 2. pp. 1–28; 217–255. [296 refs.]

This article is indeed an excellent review of a subject which has presented many difficulties and problems to which many research workers of renown have devoted much time in solving since Hansen's momentous discovery in 1874. The author considers the subject in a critical manner from the early studies of Gaucher, Thin, Steven, Lutz and Rake, who all attempted cultivation of the organism associated with lepra lesions, on the various media known in their day but were unable to obtain confirmatory results by animal inoculation. The author proceeds to describe the measures tried under periods, 1890–97, which includes the work of Hansen, Kanthack, Barclay, Ducrey, Byron, Rocca and Unna; 1897–1900 with the studies of Levy, Spronck, Czaplewski, Max Teich, Scholtz and Klingmüller; 1900–1909 mainly dealing with Kedrowski's work and that of Rost, Weil and Wherry who confirmed the pleomorphism of cultures which others had also observed; 1909–12 and the Philippine investigators, notably Clegg and the symbiotic cultivation in association with an amoeba, confirmed by Currie, Brinckerhoff and Hollmann, and the work of Teague, Duval, Serra, Bayon and others; 1913–17, the chief investigators of this period being Fraser, Fletcher, Reenstierna, McCoy and again Rost.

The question at that time may be summed up by saying that none of these organisms had established the claim to be the *true leprosy organism*. The author next considers in fuller detail the claims of Walker's diphtheroid types, the partly acid-fast organisms of Bordoni-Uffreduzzi and others, of de Souza Araujo, of Shiga, the rat-leprosy studies, and finally his own investigations with Soule. He describes his technique and the animal experiments carried out by himself and his colleague and concludes: "There seems to be no good reason to question the status of that organism [Hansen's bacillus], which is so constantly associated with lesions of this disease. Its final cultivation on artificial media will one day be accepted, another chapter in the study of this disease will be brought to a close and new vistas of study will be opened."

[It is not possible to abstract this valuable article by Dr. McKinley; it is itself a review crammed full of interesting points. The following quotation from Scott's recently published *History of Tropical Medicine* calls attention to further work on cultivation of the organism of leprosy not included in the article above. He states (p. 629):

"Throughout 1936 attempts were made at the Calcutta School of Tropical Medicine to confirm the reports of successful cultivation . . . . Twenty-four series of experiments were carried out and a thousand tubes of different media inoculated . . . . Of seventy tubes seeded in August and kept in a gaseous environment of 40 per cent. oxygen and 10 per cent. carbon dioxide, thirty-five showed slight macroscopic and considerable microscopic evidence of colony formation and many masses of acid-fast bacilli were seen in smears. Of another seventy under ordinary atmospheric conditions fourteen showed similar but less evident growth."

On the same page, referring to McKinley's own work:

"By 1937 McKinley and de Leon reported having carried the cultivation through sixty generations. The most successful medium has proved to be hormone-glycerol-agar."]

H. H. S.

Suwo (Masasue) & Kin (Shotai). Ueber kulturen von leprösen gewebe in vitro. I. Mitteilung. Gewebskulturen von hautknötchen bei menschenlepra. [**Tissue Culture of Human Leprosy Nodules.**]—*Internat. Jl. Leprosy.* Manila. 1939. Jan. Mar. Vol. 7. No. 1. pp. 57–66. With 21 figs. on 4 plates. [10 refs.]

Bacillus-containing leprous tissues have been studied in cultures. It was found that small pieces of leprosy nodules grow well in tissue culture with fibroblasts and epitheloid cells with lepra bacilli in them, the latter appear to accumulate in the fibroblasts, and lepra cells develop from the epitheloid cells with multiplication of bacilli in them and in the fluid, but without visible colonies of the organisms.

L. R.

Rousseau (Paul) & Gaugeat (M.). II. Culture du bacille de Hansen (à partir du sang de lépreux). Technique personnelle. [**Cultures of Hansen's Bacillus from the Blood of Lepers.**]—*Rev. Méd. et Hyg. Trop.* 1938. Nov.–Dec. Vol. 30. No. 6. pp. 326–332.

These workers claim to have cultivated Hansen's bacillus from both leprous lesions and from the blood of leprosy cases in Loewenstein's media. The organisms were pleomorphic and grew readily on culture media. *L. R.*

Evans (Florence L.). **Clegg's Amoeba Culture Method for growing** *Mycobacterium leprae.*—*Public Health Rep.* 1939. Feb. 24. Vol. 54. No. 8. pp. 301–305. [11 refs.]

This note reports a trial of Clegg's method of culture in symbiosis with the cholera vibrio or other bacteria and amoebae. Two chromogenic Mycobacteria were obtained which are considered to be possibly the causative organism of leprosy. *L. R.*

Oberdörffer (M.). **Introduction to an Investigation of Racial Differences in the Clinical Picture of Leprosy.**—*Leprosy Review.* 1939. Apr. Vol. 10. No. 2. pp. 112–114.

This note repeats the hypothesis in a former paper in this *Bulletin* [1938, Vol. 35, p. 882], to the effect that leprosy is predisposed to in the tropics by the use of a food plant called *Collocasia antiquorum*, which the author states to contain a highly toxic sapotoxin with a definite seasonal variation. *L. R.*

Aykroyd (W. R.) & Krishnan (B. G.). **A Diet Survey of Families with Leprosy.**—*Indian Jl. Med. Res.* 1939. Apr. Vol. 26. No. 4. pp. 897–900.

Radna (R.). Sur la bacillurie des lépreux. [**Bacilluria in Leprosy.**]—*Ann. Soc. Belge de Méd. Trop.* 1939. Mar. 31. Vol. 19. No. 1. pp. 51–53.

The elimination of lepra bacilli in the urine has been studied in view of the finding of Berny that the organisms of rat leprosy are more frequently eliminated in animals undergoing arsenical treatment. Repeated examination of the urine of patients before the administration of arsenic always proved negative, but in 9 cases being treated with

arsenic in the form of novarsenobenzol for complicating syphilis or yaws, the urine was commonly positive for lepra bacilli in lepromatous, but not in nerve cases. Such cases should therefore be treated in hospitals when possible. *L. R.*

RADNA (R.). Contribution au problème de la transmission de la lèpre. Les formes de la lèpre dans la région de Pawa et leur infectiosité. Première note : l'élimination du bacille de la lèpre. [**Transmission of Leprosy : The Elimination of the Bacilli.**]—*Ann. Soc. Belge de Méd. Trop.* 1939. Mar. 31. Vol. 19. No. 1. pp. 39–50.

This inquiry into the modes of transmission of leprosy is in agreement with the general view of the importance of the discharge of lepra bacilli from the nose and skin. Droplet infection in laryngeal cases and the escape of lepra bacilli through the genito-urinary apparatus, in the milk, tears and stools are also considered to be possible means of infection. *L. R.*

RADNA (R.). Contribution au problème de la transmission de la lèpre. Les formes de la lèpre dans la région de Pawa et leur infectiosité. Deuxième note : la transmission du bacille de Hansen. [**Transmission of Leprosy.**]—*Ann. Soc. Belge de Méd. Trop.* 1939. June 30. Vol. 19. No. 2. pp. 201–224. With 1 graph. [13 refs.]

This is an epidemiological study of the conditions of leprosy infections in a French colony. An attempt was made to discover the sources of infection in 241 cases of leprosy by house inquiries, with success in only 132, in 125 of which the transmission occurred in the family. The possibility of hereditary transmission was considered, with the conclusion that although it could not be denied, at the most it could play very little part. Contact infection is then dealt with and the dangers of contamination by dust of rooms and through smoking pipes and cigarettes is pointed out. Infection through bedbugs, flies and other insects is considered to be possible but rare. The general conclusion is that family contact constitutes the greatest danger. *L. R.*

LEISHMANIASIS.

Précis of Abstracts in this Section.

ZAMBRANO (p. 1024) adds physiological saline or skimmed goats' milk to the water of condensation of NNN medium for the cultivation of leishmania. SENEKJI (p. 1024) describes the preparation of a medium more suitable than NNN for the primary isolation of *L. tropica*.

ADLER and THEODOR (p. 1025) studied the development of *L. chagasi* in *P. papatasi* which had been infected by feeding through a membrane upon a suspension of flagellates. Passage of flagellates to the oesophagus may occur as early as 3 days after feeding.

CHUNG and FENG (p. 1025) found flagellates indistinguishable from developing leishmania in *P. chinensis*. They (p. 1025) show that *P. chinensis* is a satisfactory host for the parasite of canine kala azar in Peiping, and consider that this fact may throw light on the epidemiology of human disease if the parasites are identical.

GIRAUD *et al.* (p. 1026) restate the theory that in Marseilles kala azar is primarily a disease of dogs and that it is transmitted to children by the dog tick. SERGENT *et al.* (p. 1026) also believe that Mediterranean kala azar is mainly a disease of dogs and that transmission is from dog to dog and from dog to child rather than from child to child. They therefore advise control of dogs and prevention of contact, but they incriminate sandflies, dog ticks not being mentioned.

DA CUNHA (p. 1027) shows that in the skin of dogs infected with S. American kala azar there are collections of macrophages containing parasites. The skin may otherwise appear to be normal. He considers that this parasite is identical with *L. infantum*.

FENG *et al.* (p. 1027) describe the symptoms in 12 dogs suffering from kala azar in Peiping, and HO (p. 1027) notes that there was a child with kala azar associated with one of these dogs.

MENDES (p. 1028) records canine kala azar in Lisbon.

PAPANTONAKIS (p. 1028) describes the measures taken in Canea to prevent the spread of canine kala azar and its transmission to man. All dogs are carefully examined and the destruction of infected dogs has been followed by reduction in the number of human cases.

BLANC (p. 1028) found a squirrel dead of leishmania infection in Morocco.

MURANO and VECCHIO (p. 1029) consider the anaemia of kala azar to be due to alterations in the reticulo-endothelial system brought about by the leishmania.

MENON (p. 1029) discusses the splenic reaction in kala azar, which at first is a reticulo-endotheliosis. DE VRIES (p. 1029) regards a positive formol-gel reaction as merely an indication of hyperglobulinaemia.

GREVAL *et al.* (p. 1030) quote figures to show the value of complement fixation tests with a certain technique, using the W.K.K. antigen.

NAPIER (p. 1030) though regarding sternal puncture as a valuable aid to diagnosis, obtains better results from splenic puncture which, in his hands, has not proved harmful.

An incubation period of 1–2 years was a feature of a case reported by PALLISTER (p. 1030). Two cases of kala azar from the South of France are reported by OLMER *et al.* (p. 1030). LEAHY (p. 1031) reports kala azar, probably contracted in Sicily, in a child in the United States. SHAPIRO and BTESH (p. 1031) describe a child in Palestine with kala azar and leishmaniasis of the face. DASTIDAR (p. 1031) describes post kala azar dermal leishmaniasis in an Indian. Leishmania were found in nodules on the tongue. LINTON (p. 1031) describes kala azar in a Lascar seaman in Glasgow. GATTI *et al.* (p. 1032) describe kala azar in a Paraguayan, apparently contracted in Bolivia.

FERRABOUC *et al.* (p. 1032) describe clinical cases in one of which the early stages were afebrile, with consequent delay in diagnosis.

ERMEN (p. 1032) shows that neostibosan produces histiocyte response and leucocytosis in hamsters, but fouadin a leucopenia. WANG (p. 1032) found a higher rate of cure in infected hamsters with neostibosan than with solustibosan. Daily injections are less effective with both than bi-weekly or tri-weekly injections.

AVERSA (p. 1033) speaks well of solustibosan in infantile kala azar. MIRZOJAN (p. 1033) reports on the value of sourmine (stibosan) in treatment. DAS GUPTA (p. 1033) shows that allergic symptoms after intravenous ureastibamine, once common, have not often been seen since smaller (but still efficacious) dosage was adopted.

Poggi and Monti (p. 1033) show that oriental sore is common in the province of Forli, Italy. Vanni (p. 1034) incriminates *P. macedonicus* as the intermediate host of *L. tropica* in Abruzzi.

Jerace (p. 1034) shows that in Teramo (Italy) the distribution of *P. perfiliewi* corresponds with that of cutaneous leishmaniasis. Berberian (p. 1034) has succeeded in effecting further transmissions of oriental sore by allowing *Stomoxys calcitrans* to bite volunteers immediately after feeding on a sore. He (p. 1035) succeeded in infecting 35 volunteers with *L. tropica* from cultures and considers this procedure to be useful in conferring immunity.

Pellettieri (p. 1035) reports oriental sore in a patient from Eritrea.

da Cunha and Dias (p. 1035) use an antigen prepared from strains of leishmania isolated from S. American cutaneous leishmaniasis.

Pifano (p. 1035) finds fouadin to be the treatment of choice for muco-cutaneous leishmaniasis in the Argentine.

Weinman (p. 1036) describes a technique for the cultivation of *L. tropica* in pieces of guineapig tissue on the surface of blood or serum-Tyrode agar. *C. W.*

Zambrano (Elio). Coltivabilità del parassita di Leishman su terreno NNN con aggiunta di soluzione fisiologica clorosodica. [**Culturability of the Leishmania Parasite on NNN Medium with Added Physiological Saline Solution.**]—*Giorn. di Batteriol. e Immunol.* 1939. June. Vol. 22. No. 6. pp. 881–890. [10 refs.] English summary (9 lines).

The author shows that leishmania can be successfully cultured and subcultured in NNN medium in which the water of condensation has been replaced or increased by the addition of physiological saline solution. Before inoculation the liquid is allowed to remain in contact with the surface of the blood agar for 24 hours in the incubator at 37°C. Equally good results are obtained by the similar use of skimmed goats' milk. *C. M. Wenyon.*

Senekji (H. A.). **Studies on the Culture of** *Leishmania tropica.*—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1939. July 28. Vol. 33. No. 2. pp. 267–269.

The author has found that ordinary NNN medium does not give very satisfactory results for the primary isolation of *Leishmania tropica* from oriental sores. A better medium is prepared as follows. To 50 grammes of Bacto beef, 1,000 cc. of distilled water is added. The mixture is infused for one hour at 50°C. and then heated for five minutes at 80°C. to coagulate some of the proteid. After keeping in the ice-chest for 24 hours the mixture is filtered. To the filtrate are then added neopeptone (Difco), 20 grammes; sodium chloride 5 grammes; agar (Nobel) 20 grammes. When solution has taken place by heating, the *p*H is adjusted to 7·2 to 7·4, and 1·5 cc. amounts are distributed in test tubes and 30 cc. amounts in Blake bottles with screw caps. After the tubes and bottles have been sterilized in the autoclave they are cooled to 55°C., at which temperature defibrinated rabbit blood is added to an amount equal to 15 per cent. of the medium. After gently mixing the contents, the tubes and bottles are sloped. Primary cultures are readily obtained in the water of condensation

of the tubes. By inoculating the water of condensation in the bottles with cultures and allowing the water to flood the agar, surface cultures are obtained. *C. M. W.*

Adler (S.) & Theodor (O.). **The Behaviour of *Leishmania chagasi* in *Phlebotomus papatasii*.—*Ann. Trop. Med. & Parasit.* 1939. Mar. 31. Vol. 33. No. 1. pp. 45–47.

The authors have already described a method for inducing sandflies to feed through a membrane on suspensions of leishmania flagellates. In the case of *Phlebotomus papatasi* the flies feed with reluctance unless the fluid contains red blood corpuscles. If, however, the membrane is smeared with a solution of cane sugar, as many as 80 per cent. of the flies will feed. The relative infectivity of different flies can be tested by allowing them to ingest emulsions of known flagellate content. Generally it is found that the percentage of flies becoming infected increases with the increase in the flagellate content of the emulsion. Thus of flies fed on emulsions of *Leishmania chagasi* containing 300 flagellates per 0·1 cmm. 24 per cent. became infected, while of those fed on emulsions containing 1,000–2,000 per 0·1 cmm. 71 per cent. were infected, while with still higher concentrations the figure was 89 per cent. When an infection has been established in the flies, the flagellates multiply in the midgut and ascend to the anterior part of the cardia which may become choked with flagellates. Passage into the oesophagus may occur as early as three days after feeding. In its behaviour in *P. papatasi*, *L. chagasi* resembles *L. donovani* and *L. infantum*, since both these give a low infection rate in flies fed on emulsions containing 300–400 flagellates per 0·1 cmm. of emulsion.

C. M. W.

Chung (Huei-Lan) & Feng (Lan-Chou). **Natural Infection of *Phlebotomus chinensis* in Peiping with Leishmania Flagellates.**—*Chinese Med. Jl.* 1939. July. Vol. 56. No. 1. pp. 47–51. With 1 plate.

In the kennel of a dog suffering from canine kala azar and in the house of the owner in Peiping, 16 specimens of *Phlebotomus chinensis* were captured between the 5th and 10th June, 1939. Of these flies two were found to be infected with flagellates indistinguishable from developmental forms of leishmania. *C. M. W.*

Feng (Lan-Chou) & Chung (Huei-Lan). **The Development of Leishmania in Chinese Sandflies fed on Dogs with Canine Leishmaniasis.**—*Chinese Med. Jl.* 1939. July. Vol. 56. No. 1. pp. 35–46. With 5 figs. on 2 plates. [12 refs.]

The authors in Peiping, N. China, have fed sandflies on dogs naturally or experimentally infected with canine leishmaniasis. Two species of sandfly were used, *Phlebotomus sergenti* var. *mongolensis* and *P. chinensis*. Both became infected with flagellates, but the latter more readily than the former, which lost its infection when the blood meal was completely digested. In *P. chinensis* the infection persisted and extended forwards to the pharynx and proboscis, a feature which was not observed in the transitory infection in *P. sergenti*. In the case of both sandflies, flagellates were found in the hind gut, while

they were seen in faeces deposited by one *P. chinensis*. The rate of infection of the sandflies was directly proportional to the degree of infection of the skin of the dogs. One dog with a heavy skin infection led to the infection of the majority of both species of sandfly fed on it. The results with the canine disease agree fairly closely with those previously obtained in the case of human kala azar by Young and Hertig, 1926, Patton and Hindle 1927, Hindle 1928 and Wu and Sun 1938, who concluded that *P. chinensis* was the host for the parasite of human kala azar. The present finding that this sandfly is a satisfactory host for the parasite of canine kala azar affords an explanation of the frequent occurrence of the canine disease in Peiping, and may also throw light on the epidemiology of human kala azar should it be proved that the human and canine diseases in China are identical. [See also this *Bulletin*, 1927, Vol. 24, pp. 133, 639; 1929, Vol. 26, p. 311; 1938, Vol. 35, pp. 861, 862.] *C. M. W.*

Giraud (Paul) & Bergier (Pierre). Epidémiologie de la leishmaniose humaine à Marseille. [**Epidemiology of Human Kala Azar in Marseilles.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris*. 1939. Apr. 10. 55th Year. 3rd Ser. No. 12. 588–591. With 1 chart.

—— & ——. Recherches sur le mode de transmission de la leishmaniose dans la région marseillaise. [**Mode of Transmission of Kala Azar in the Marseilles Region.**]—*Ibid.* pp. 591–593.

——, Cabassu (H.) & Bergier (Pierre). Recherches sur l'endémie de leishmaniose canine à Marseille. [**Endemic Canine Kala Azar in Marseilles.**]—*Ibid.* pp. 594–596. With 1 chart.

In these three papers the epidemiology of kala azar in Marseilles is discussed. In human beings the disease most commonly attacks children between the ages of one and three. By far the greater proportion of cases comes from the districts round Marseilles itself or from houses in the suburbs where there are gardens. Canine kala azar is also common and is found chiefly in the same districts as the human cases. The conclusion is that kala azar is essentially a canine disease which passes to human beings accidentally. Of vectors, the authors suspect the dog tick, which is found in greatest numbers on the types of dog most subject to kala azar, namely hunting dogs and police dogs. The dog tick bites children which play about on the ground and come into closer contact with dogs than do adults, amongst whom the disease is rarely found. The sandfly bites adults, as frequently as children, and dogs less often. One case is mentioned in which a child suffered from the tick-borne fièvre boutonneuse in September and kala azar in November of the same year. The question is asked, was this an instance of simultaneous inoculation of two viruses? The possibility of the dog louse (*Linognathus piliferus*) being a vector is put aside, in spite of the fact that Professor Joyeux is stated to have shown that leishmania will develop in it. *C. M. W.*

Sergent (Edm.), Parrot (L.), Donatien (A.) & Lestoquard (F.). La prophylaxie de la leishmaniose générale méditerranéenne. [**Prophylaxis of Mediterranean Kala Azar.**]—*Arch. Inst. Pasteur d'Algérie*. 1939. June. Vol. 17. No. 2. pp. 221–230.

The paper discusses the prophylaxis of Mediterranean kala azar, which occurs in human beings and dogs and appears to be transmitted by sandflies, chiefly *Phlebotomus perniciosus*, from dog to dog and

from dog to human being rather than from one human being to another. In practically all centres of the disease there are more cases amongst dogs than human beings, while sandflies are much more readily infected by feeding on dogs, owing to the tendency in these animals for the parasites to be present in large numbers in the skin [see DA CUNHA below]. It has been found that reduction in the human reservoir by treatment of all cases has not been followed by a lowered incidence of the disease. As the disease mostly affects children, contact of these with dogs should be prevented. All known infected dogs and all stray unowned dogs should be destroyed. The movement of dogs into and from known centres of the disease should be forbidden, it being borne in mind that many dogs harbour parasites without showing any signs of their infection. As regards sandflies, protection from bites can be largely effected by the use of a sufficiently fine net at nights, while destruction of flies in the rooms can be carried out. Breeding can be prevented to some extent by working the land round the house, burying accumulations of rubbish and generally keeping the surroundings clean. *C. M. W.*

DA CUNHA (Aristides Marques). Infecção da pelle na leishmaniose visceral experimental do cão. [**Condition of the Skin in Dogs experimentally infected with Kala Azar.**]—*Brasil-Medico.* 1938. Nov. 26. Vol. 52. No. 48. pp. 1071–1072.

A study of the skin of dogs experimentally infected with the leishmania of S. American kala azar has shown that, as in the case of dogs in the Mediterranean region, it becomes infiltrated with macrophages containing parasites. The skin, apart from the presence of macrophages, may be quite normal and have no visible signs of infection, or there may develop areas of tumefaction in which numerous leishmania can be found. The observations, like others that have been made before, lend support to the view that the parasite of S. American visceral leishmaniasis is identical with *Leishmania infantum.* *C. M. W.*

FENG (L. C.), CHUNG (H. L.) & HOEPPLI (R.). **Canine Leishmaniasis with Skin Lesions observed in Peiping.**—*Chinese Med. Jl.* 1939. Apr. Vol. 55. No. 4. pp. 371–382. With 15 figs. on 4 plates.

The paper gives an account of twelve dogs which were found to be suffering from canine leishmaniasis in Peiping. In all cases diagnosis was established by the finding of leishmania in smears from cutaneous lesions. The symptoms in these dogs were seborrhea, scaling and depilation of the skin, small nodules, ulcerations on various parts of the body but especially on the ears, face, nose and round the eyes. In two dogs keratitis was noted and marked emaciation in five. The other dogs appeared quite healthy apart from the skin lesions. In none of these cases was examination of the internal organs carried out, but there is little doubt that the dogs were suffering from canine kala azar. *C. M. W.*

HO (E. A.). **A Note on the Coincidence of Human Kala-Azar and Canine Leishmaniasis with Cutaneous Lesions in a Household.**—*Chinese Med. Jl.* 1939. June. Vol. 55. No. 6. pp. 566–567.

The author notes that one of the twelve dogs reported as suffering from canine leishmaniasis in another paper reviewed here [FENG,

Chung and Hoeppli above] came from a house in which a case of kala azar in a child 2½ years of age occurred. The dog had commenced to show skin lesions as evidence of its infection about a year before the appearance of the first symptoms in the child. A similar association of canine and infantile kala azar in a house in Peiping has been reported by Lee in 1937. *C. M. W.*

Mendes (A. B. Corrêa). Leishmaniose nos cães de Lisboa. [**Leishmaniasis in Lisbon Dogs.**]—*Arquivos Inst. Bact. Camara Pestana.* 1938. Vol. 7. No. 3. pp. 389–395. English summary.

In 1911, Alvares and Pereira da Silva reported the finding in Lisbon of 13 cases of canine kala azar amongst 416 dogs examined. In the present paper the author records the discovery of leishmania in 5 of 240 dogs examined post-mortem. In all cases but one the presence of parasites was associated with a positive formol-gel reaction, but in three cases giving a positive reaction leishmania were not found, though two of these were instances of infection with *Dirofilaria immitis* and one with *Babesia canis.* *C. M. W.*

Papantonakis (Evangelos). Bekämpfungsmassnahmen der Kala-Azar in Canea/Kreta. [**Kala Azar Control Measures in Canea (Crete).**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1939. June. Vol. 43. No. 6. pp. 273–275. With 1 fig.

The close association of human and canine kala azar in Canea in Crete has strengthened the belief that the dog is acting there as the reservoir of infection which is transmitted to human beings by sandflies. The destruction of the majority of the dogs in the district of Canea in 1933 was followed by a markedly lower incidence of human kala azar in the following year. Accordingly regulations were drawn up by the Health Department insisting on an inspection of all dogs of Canea in April of each year. Each dog was examined clinically as well as serologically by the formol-gel test. All sick dogs and those giving a positive test were killed. Furthermore, if in any village of the district a case of human kala azar occurs, all the dogs of the village are to be at once destroyed. Anyone who objects to his dog being killed may take it for test to the Hygiene Centre in the town as in the case of the town dogs. The residents of such a purged village can only acquire new dogs in the months of January or February, while the animals must be under two months of age and must have come from villages where no cases of the human disease have been reported.

The general inspection of dogs in Canea was first carried out in April 1938. Of 1,115 dogs 229 gave a positive formol-gel test. Of these 70 per cent. showed no signs of disease, 25 per cent. merely some wasting and loss of hair and only 5 per cent. the characteristic signs of canine kala azar. *C. M. W.*

Blanc (G.). Leishmaniose viscérale généralisée observée chez un écureuil (*Xerus getulus* L.) au Maroc. [**Generalized Kala Azar in a Squirrel (*Xerus getulus*) in Morocco.**]—*Bull. Soc. Path. Exot.* 1939. July 12. Vol. 32. No. 7. pp. 756–761. With 2 plates. [12 refs.]

Among a number of squirrels (*Xerus getulus*) bred at the Pasteur Institute in Morocco one was discovered to have died of a very intense

leishmania infection. The origin of the infection could not be traced as no animals with this infection had been housed in the building for some time. It is admitted that dogs and possibly other animals may harbour leishmania though they appear quite healthy. Furthermore, it was not possible to identify the species of parasite as the infection was not transmissible to other animals owing to the decomposed state of the body of the squirrel. The possibility of this animal being a reservoir for leishmania infection is discussed, though it is admitted that the only conclusion that can be drawn from the facts is that it is susceptible to infection. *C. M. W.*

Murano (Giulio) & Vecchio (Federico). Sull'interessamento del sistema reticolo-istiocitario nella leishmaniosi interna. I. L'anemia da leishmaniosi interna. [**Relation of the Reticulo-Endothelial System to Kala Azar. I. The Anaemia of Kala Azar.**]—*Pediatria.* 1939. July. Vol. 47. No. 7. pp. 545–563. [43 refs.]

An examination of the blood of a number of cases of infantile kala azar in Naples and the study of rabbits by blockages of the reticulo-endothelial system has led the authors to the view that the anaemia of kala azar cannot be attributed to any haemolytic process. It is due rather to a hyperplasia of the bone marrow caused by alterations in the reticulo-endothelial system brought about by the leishmania.

C. M. W.

Menon (T. Bhaskara). **The Splenic Reaction in Kala-Azar.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1939. June 29. Vol. 33. No. 1. pp. 75–86. With 8 figs. on 4 plates. [47 refs.]

This study, based on material from ten fatal cases of kala azar, has shown that the disease is a reticulo-endotheliosis caused by a parasitic invasion of the cytoplasmic reticular syncytium. There is a gradual differentiation of the reticulo-endothelial tissue to form free histiocytes, which are also parasitized. The splenic reaction affords suggestive evidence for the presence in the spleen of two types of phagocytic cell with differing powers of phagocytosis. In the later stages there is a spread of infection to the lymphatic reticulum of the spleen. Such an activation and invasion of the lymphatic reticulum is offered as the explanation of the involvement of the lymphatic tissue in the disease. Reticulo-endothelial " blockage " is regarded as the probable cause of the anaemia and tendency to inflammatory complications.

C. M. W.

de Vries (A.). **On the Connection between the Formol-gel Reaction and the Blood Proteins.**—*Acta Med. Scandinavica.* 1939. Vol. 99. No. 5. pp. 425–434. With 1 graph. [11 refs.]

In this paper the author gives an account of his examination of the serum from a large number of different diseases. He has found that whenever the total globulin content reached 37 parts per thousand the formol-gel reaction was positive, while it was uniformly negative when the value fell to below 36 parts per thousand. Between 36 and 37 the reaction was either positive or negative. A decrease in the albumin content had little influence on the reaction. It is clear

that the reaction is not specific for any particular disease or group of diseases, but that it is merely an indication of a hyperglobulinaemia.

C. M. W.

Greval (S. D. S.), Sen Gupta (P. C.) & Napier (L. Everard). **Serological Reactions in Kala-Azar : Complement-Fixation, False Wassermann Reaction, and High Anti-Complementary Titre.**—*Indian Jl. Med. Res.* 1939. July. Vol. 27. No. 1. pp. 181–190.

The paper describes a technique of complement fixation with W.K.K. (Witebsky-Klingenstein-Kuhn) antigen in kala azar in Calcutta. The haemolytic system of method No. 4 of the Wassermann test of the British Medical Research Committee, a maximum quantity of the antigen which does not interfere with 1 m.h.d. of complement, and a 1 in 25 or more dilution of the serum to be tested are required. The diagnostic value of the reaction is high, for of 132 cases it was found that not a single diagnosed case of kala azar gave a negative reaction. Six cases were doubtful or negative with the formaldehyde test but fully positive with the reaction. All were later proved to be cases of kala azar by discovery of parasites. *C. M. W.*

Napier (L. Everard). **Sternal Puncture in Kala-Azar.** [Correspondence.]—*Lancet.* 1939. Apr. 22. p. 959.

Writing of sternal puncture as a procedure for the diagnosis of kala azar the author, who has had a very wide experience of splenic puncture for this purpose, concludes that sternal puncture has given another fairly accurate means of making a diagnosis, but that it has not yet been proven that it is as reliable a method as splenic puncture. In a series of sternal punctures in cases of kala azar negative findings were the result in 20 per cent. of the cases. Of the negative cases, 11 in all, parasites were discovered in 9 by splenic puncture. A long experience has shown the author that splenic puncture in kala azar cases will yield parasites in about 95 per cent. of cases. Another point is that the parasites are much less numerous in a film of bone marrow than they are in a film of spleen material. As regards the dangers of splenic puncture, the author states, as he has done before, that during 15 years he has performed well over 5,000 splenic punctures without any serious effects. *C. M. W.*

Pallister (R. A.). **A Case of Kala-Azar.**—*Jl. Malaya Branch Brit. Med. Assoc.* 1939. Mar. Vol. 2. No. 4. pp. 250–251. With 1 fig.

The case reported is that of an Indian in Malaya, who apparently contracted the disease during a visit to India from one to two years before he reported sick at the General Hospital, Penang, where the disease was diagnosed. The case is remarkable for the length of the incubation period. The disease was brought under control by antimony treatment. *C. M. W.*

Olmer (Jean), Montagnier (L.) & Mingardon (G.). Remarques à propos de deux nouveaux cas de kala-azar de l'adulte. [**Two Cases of Kala Azar in Adults.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1939. May 8. 55th Year. 3rd Ser. No. 14. pp. 682–685.

The paper reports two cases of kala azar in adults in the south of France, the one being sixty years of age and the other forty-four. The

first case was treated by twelve injections of neostibosan followed by twelve of stibyal given every two or three days, while the second was given eighteen injections of neostibosan during the course of three weeks. The treatment produced no unpleasant results and appears to have cured both cases. *C. M. W.*

Leahy (Hugh F.). **Kala-Azar.**—*Amer. Jl. Dis. Children.* 1939. May. Vol. 57. No. 5. pp. 1085–1098. With 5 figs. [13 refs.]

The case reported is that of a child thirteen months old who was found to be suffering from kala azar in the United States, the disease having been contracted in Sicily where the child was born of Italian parents. Treatment with fouadin seems to have controlled the disease, but death resulted from intercurrent infections. *C. M. W.*

Shapiro (J. M.) & Btesh (S.). **Kala-Azar in Palestine, with Special Reference to a Case with Cutaneous Lesions.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1939. July 28. Vol. 33. No. 2. pp. 257–262. With 2 figs. on 1 plate.

In giving an account of eight cases of infantile kala azar admitted to the Government Hospital at Haifa during the year March 1938 to April 1939, particular attention is called to one case in which in an area of about an inch in diameter on one cheek there were five small papules. Material both from the spleen and papules showed leishmania. As both oriental sore and kala-azar occur in the district, the question whether the skin condition was a manifestation of kala azar or oriental sore could not be settled. It is noted that every few days there was a skin reaction around the papules accompanied by a rise in temperature. Adler is quoted as saying that since 1920 there have been twenty-five cases of kala azar recognized in Palestine, all of which have been in children, except two cases in adults. *C. M. W.*

Dastidar (S. K. Ghosh). **A Rare Tumour of the Tongue. A Manifestation of Post-Kala-Azar Dermal Leishmaniasis.**—*Brit. Med. Jl.* 1939. July 1. pp. 14–15.

The case described is that of an Indian at Patna who developed post-kala azar dermal leishmaniasis following incompletely treated kala azar. Apart from nodules and depigmented spots on the skin of the face, neck and upper part of the chest, there developed a nodular condition of the tongue. In material obtained from one of the lingual nodules, leishmania were found. Though lesions of the tongue have been twice described in this condition, this is the first time that parasites have actually been demonstrated in nodules on the tongue.
C. M. W.

Linton (W. S.). **A Case of Kala-Azar.** [Memoranda.]—*Brit. Med. Jl.* 1939. Aug. 12. p. 336.

The case reported is that of a Lascar seaman who was found to be suffering from kala azar after admission to hospital in Glasgow by the discovery of leishmania within a polymorphonuclear leucocyte in a film of the peripheral blood, which was being searched for malarial parasites. There was a rapid response to treatment with potassium antimony tartrate. *C. M. W.*

Gatti (C.), Boggino (J.) & Prieto (C.). Un nouveau foyer de leishmaniose viscérale en Amérique du Sud. [**A New Focus of Kala Azar in S. America.**]—*Bull. Soc. Path. Exot.* 1939. June 14. Vol. 32. No. 6. pp. 602–605. With 1 fig.

The paper describes a case of kala azar in an adult Paraguayan. Though he had travelled, a residence of a year in Yungas in the north of Bolivia before the first symptoms of infection developed appears to indicate that this district must be added to those already recognized as centres of the disease in South America. The other centres are those of Mattogrosso on the borders of Brazil and Paraguay, those of Salta and Chaco in Northern Argentine and that of North-Eastern Brazil, south and east of Para. *C. M. W.*

Ferrabouc (L.), Jaulmes (C.) & Jude (A.). Remarques cliniques sur deux cas de kala-azar. [**Clinical Observations on Two Cases of Kala Azar.**]—*Rev. Méd. et Hyg. Trop.* 1939. Mar.–Apr. Vol. 31. No. 2. pp. 56–61.

Two cases of kala azar are described in adults who had returned to France from Morocco and French Somaliland. It seems probable, though not quite certain, that the infections had been contracted in these countries. In one case the diagnosis was difficult owing to the main symptoms resembling those of acute peritonitis. In the other the early stages of the disease were afebrile, the patient being admitted to hospital for general debility combined with enlargement of the liver and spleen. A diagnosis of cirrhosis was made. It was not till six months later that the first febrile symptoms developed. The first case was much more resistant to antimony treatment than the second, several courses of large dose being required to bring about a cure. *C. M. W.*

Ermen (Johannes). Die Wirkung von 3- und 5wertigem Antimon auf das weisse Blutbild bei gesunden und mit Kala-azar infizierten Hamstern. [**The Influence of Tri- and Pentavalent Antimonials on the Leucocytes of the Blood in Healthy and Kala Azar Infected Hamsters.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1938. July 8. Vol. 93. No. 3/4. pp. 209–228. With 5 figs. & 7 charts. [22 refs.]

This paper has already been the subject of a review [this *Bulletin*, 1938, Vol. 35, p. 870] but it was there erroneously stated that a leucocytosis followed the administration of fouadin to healthy hamsters, whereas the reverse is the case. It thus appears that neostibosan administered to hamsters produces a marked histiocyte response as well as an increase in the number of leucocytes, an action which is related to the therapeutic efficiency of the drug in the treatment of leishmania infections in these animals. Fouadin not effective for treatment produces only a very slight histiocyte response and a decrease in the number of leucocytes. *C. M. W.*

Wang (C. W.). **Solustibosan in Treatment of Kala-Azar in Chinese Hamsters.**—*Proc. Soc. Experim. Biol. & Med.* 1939. May. Vol. 41. No. 1. pp. 152–155.

The authors have found that in the treatment of Chinese hamsters, daily subcutaneous injections of solustibosan (120 mgm. of Sb) for

10 to 25 days showed a lower cure rate than neostibosan (168 mgm. of Sb) given in the same way over the same period. Furthermore, the curative effect of solustibosan did not increase with the increase of the frequency of injections. Daily injections of either drug appear to be less effective than bi-weekly or tri-weekly injections. This result probably depends on the fact, demonstrated by BRAHMACHARI, that when the antimony in the tissues reaches a certain level the excretion by the kidneys becomes suddenly increased out of all proportion to the antimony in the tissues. *C. M. W.*

AVERSA (Tommaso). Primi tentativi di cura della leishmaniosi viscerale infantile col solustibosan. (Nota preventiva.) [**Treatment of Infantile Kala Azar with Solustibosan.**]—*Pediatria.* 1939. June. Vol. 47. No. 6. pp. 486–489.

Very good results have followed the use of solustibosan in the treatment of five cases of infantile kala azar in Messina. The preparation was better tolerated than other organic antimonials.

C. M. W.

MIRZOJAN (N. A.). Traitement de la leischmaniose viscérale par la sourmine. [**Treatment of Kala Azar by Sourmine.**]—*Med. Parasit & Parasitic Dis.* Moscow. 1938. Vol. 7. No. 4. [In Russian pp. 596–605. [15 refs.] French summary p. 605.]

The paper reports that the synthetic organic antimony preparation named sourmine and prepared in the USSR is very efficacious in the treatment of visceral leishmaniasis in Samarkand.

According to the constitutional formula given in the text "sourmine" is the sodium salt of 3-chloro-4-acetylaminophenylstibonic acid, *i.e.*, it is identical with the German drug "stibosan," which was the first improvement on stibacetin and was discarded in favour of von Heyden 693, which was in turn replaced by "neostibosan."

C. M. W.

DAS GUPTA (N. C.). **Allergic (?) Symptoms in Cases of Kala-Azar treated with Antimony Salts.**—*Jl. Indian Med. Assoc.* 1939. Jan. Vol. 8. No. 4. p. 237.

Two cases are mentioned in which allergic symptoms developed shortly after the intravenous injection of a dose of ureastibamine for the treatment of kala azar. In both cases there was the development over the body of an urticaria-like eruption. One patient became unconscious while the other developed uterine haemorrhage. The author says that such cases were common in the early days of ureastibamine but since smaller doses (0·01 to 0·10 gm.) have been given the allergic symptoms have not been seen, while the treatment has been quite effective. *C. M. W.*

POGGI (Igino) & MONTI (Giuseppe). Esteso focolaio endemico di leishmaniosi cutanea in Provincia di Forli. [**Large Endemic Focus of Oriental Sore in the Province of Forli.**]—*Ann. d'Igiene.* 1939. June. Vol. 49. No. 6. pp. 375–379. With 2 figs. & 1 map.

The authors have shown that oriental sore is common in the villages of the valley of the Conca in the province of Forli, in Italy. It appears

also to be prevalent in the neighbouring valleys of the Marecchia and the Foglia, all of which rivers discharge into the Adriatic.

C. M. W.

Vanni (Vittorio). Ricerche sulla leishmaniosi cutanea endemica degli Abruzzi. (Transmissione sperimentale). [**The Endemic Kala Azar of the Abruzzi.**]—*Ann. d'Igiene.* 1939. Feb. Vol. 49. No. 2. pp. 65–67. [11 refs.]

Working in Abruzzi, in Italy, where oriental sore is endemic, the author has found, among 1,600 *Phlebotomus macedonicus* examined, one with a pharyngeal leptomonad infection. Inoculation of an emulsion of 200 of these sandflies into the root of the tail of three rats resulted in the development of a lesion containing leishmania in one of the animals after an incubation period of 40 days. It would seem that the sandfly *P. macedonicus* is the intermediate host for *Leishmania tropica* in Abruzzi. *C. M. W.*

Jerace (Felice). I flebotomi degli Abruzzi. IIIa nota. (Distribuzione e biologia dei flebotomi in prov. di Teramo.) [**Distribution and Biology of** *Phlebotomus* **in the Province of Teramo in Abruzzi.**] —*Ann. d'Igiene.* 1939. May. Vol. 49. No. 5. pp. 309–315. With 1 fig.

Three species of *Phlebotomus* were found in the province of Teramo—*P. perfiliewi*, *P. papatasi* and *P. perniciosus*. The distribution of these is shown on a sketch map from which it appears that the distribution of *P. perfiliewi* coincides with that of cutaneous leishmaniasis. This bears out the finding by Vanni in this district of a Phlebotomus naturally infected with flagellates. The author also gives a brief account of the biology and habits of *P. major*, which he was the first to discover in Abruzzi, and summarizes the technique for rearing these insects and for studying their morphology.

V. B. Wigglesworth.

Berberian (D. A.). **A Second Note on Successful Transmission of Oriental Sore by the Bites of** *Stomoxys calcitrans*.—*Ann. Trop. Med. & Parasit.* 1939. Mar. 31. Vol. 33. No. 1. pp. 95–96. With 3 figs.

In a previous paper the author has described experiments in which transmission of oriental sore from one person to another was effected by feeding *Stomoxys calcitrans* on a sore and allowing them to feed immediately on a volunteer [see this *Bulletin*, 1938, Vol. 35, p. 863.] After an incubation period of five months two sores developed. The experiment has now been repeated by allowing three of these flies to feed on one of the sores which developed on the volunteer. The flies fed on the sore ten times and on the thigh of a volunteer five times. Here again, and after a similar incubation period, two sores developed. Thus four successful mechanical transmissions of oriental sore have been made by means of *Stomoxys calcitrans*. *C. M. W.*

BERBERIAN (D. A.). **Vaccination and Immunity against Oriental Sore.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1939. June 29. Vol. 33. No. 1. pp. 87–94. With 5 figs. on 1 plate.

It is recorded in this paper that the author made forty-four inoculations of cultures of *Leishmania tropica* in thirty-five volunteers. The quantity of inoculum varied from 0·05 to 0·2 cc. according to the richness of the cultures, which were 4 to 50 days old first, second, third or fourth generation cultures. The site chosen was the skin of the thigh. In all, thirty-five sores developed. It is concluded that the procedure is a simple one for conferring immunity against the development of oriental sore on exposed parts of the body. It is noted that inoculations of culture into individuals already immune is followed by a definite skin reaction, both immediate and delayed.

C. M. W.

PELLETTIERI (Luigi). Un caso di leishmaniosi cutanea osservato in un lavoratore reduce dall' A.O.I. [**Cutaneous Leishmaniasis in a Labourer discharged from Italian East Africa.**]—*Riforma Med.* 1939. May 27. Vol. 55. No. 21. pp. 800, 803–806. With 2 figs.

The case reported is that of a labourer who contracted oriental sore during a stay in Eritrea. The sore was a lesion on the lobe of the ear which, in Africa, proved very refractory to injections of neostibosan. Later when the treatment was repeated in Italy, a good response occurred owing no doubt to the better conditions of life and health obtaining there. *C. M. W.*

DA CUNHA (A. Marques) & DIAS (Emmanuel). Sur la préparation d'un antigène stable pour la réaction de fixation du complément dans les leishmanioses. [**A Stable Antigen for Fixation of Complement in the Leishmaniases.**]—*C. R. Soc. Biol.* 1938. Vol. 129. No. 33. pp. 991–993.

This appears to be another version of the paper reviewed in this *Bulletin*, 1939, Vol. 36, p. 451. The antigen has been prepared from a number of strains of leishmania both from visceral and cutaneous conditions from both the Old and New Worlds. The most active antigen was prepared from a strain isolated from a case of S. American cutaneous leishmaniasis. Very definite positive reactions have been obtained with sera from visceral and cutaneous leishmaniasis occurring naturally in human beings and both naturally and experimentally in animals. *C. M. W.*

PIFANO (Felix). Contribución al estudio epidemiologico y clinico de la leishmaniasis cutaneo-mucosa en el estado Yaracuy. [**Epidemiological and Clinical Study of Muco-cutaneous Leishmaniasis in the State of Yaracuy.**]—*Bol. Ministerio de Sanidad y Asistencia Social.* Caracas. 1939. Mar. Vol. 4. No. 2. pp. 7–15. [27 refs.]

The author reports that during three years residence in the state of Yaracuy in the Argentine he has encountered thirty-six cases of cutaneous or muco-cutaneous leishmaniasis. The majority, 26, had cutaneous lesions only, eight had muco-cutaneous lesions, while

in two there were mucosal lesions only. Fouadin administered intramuscularly appears to be the treatment of choice for these conditions. The recent discovery in Brazil and northern Argentine of cases of visceral leishmaniasis leads the author to suspect that cases will be discovered in Yaracuy. He recalls that the first case of kala azar in S. America to be recorded was one seen by MIGONE in 1913 in Asuncion, while two cases were noted by MAZZA in 1926 in the Argentine.

C. M. W.

WEINMAN (David). **Factors affecting the Morphology of *Leishmania tropica*. The Production of *Leishmania* Forms in Cultures.**—*Parasitology*. 1939. July. Vol. 31. No. 2. pp. 185–192. [16 refs.]

The paper describes experiments which have shown that under certain conditions of culture the flagellate forms of leishmania will be transformed into the tissue-invading leishmania forms. Though the guineapig is refractory to inoculation with leishmania and though the parasite will not develop in guineapig tissue cultures, it will multiply in pieces of guineapig tissue (spleen or lung) which are placed on the surface of the NNN blood agar or Zinsser's serum-Tyrode agar. The pieces of tissue, about 2·5 mm. in diameter, are removed from the animal aseptically, washed in Tyrode's solution and then bathed in rich culture from tubes of NNN medium for 15 to 30 minutes. The pieces of tissue are then placed on the agar slopes and incubated at 33° to 37°C. The cells of the tissue appear to survive for eleven days or longer during which living leptomonads live and multiply in the drop of the liquid which collects round each piece of tissue, while in the tissue itself typical leishmania forms appear and multiply. The best results were obtained with guineapig lung, though the spleen of this animal and even that of the frog behaved similarly. The serum-Tyrode-agar was prepared as follows. To 150 cc. of 3 per cent. agar at 50°C. is added a mixture (warmed to 30°C.) consisting of 150 cc. of sterile double strength Tyrode solution, 100 cc. of horse serum and 8 cc. of 0·04 per cent. aqueous solution of phenol red. After mixing the medium is tubed and slanted. After sterility has been proved by incubation the cotton plugs are replaced by rubber caps and the tubes stored at 8°–10°C. Tubes three months old have given satisfactory results.

C. M. W.

ENQUIRIES.

To the Director,
Bureau of Hygiene & Tropical Diseases.

Sir,

In regard to your correspondent's request [this *Bulletin*, 1939, Vol. 36, p. 789] for any reference to viability of helminth eggs in sewage after tank treatment, it is but just to Ch. Wardell STILES to point out that evidence for such destruction in household septic tanks goes back to his pioneer work published in 1911. In his first Annual Report as Scientific Secretary of the Rockefeller Sanitary Commission for the Eradication of Hookworm Disease, he reported that when faecal material was subjected to decomposition in water for 70 days nearly all, and when so subjected for 117 days all, hookworm eggs were dead; and in these conditions, at least 80 per cent. of Ascaris eggs were dead in four months.

These observations were made to test the value of the L.R.S. (Lumsden, Roberts, Stiles) privy designed for one country family and described in U.S. *Public Health Reports* 1910, Nov. 11; another description of it followed in the *Farmers' Bulletin 463 of the U.S. Department of Agriculture*, 1911; while that of a modification in concrete appeared in *Public Health Bulletin No. 68* issued in 1917 by the Treasury Department, U.S. Public Health Service. On the L.R.S. privy was based the Aqua privy, a factory-made concrete cylinder with drowned, unscreened outlet [LANE, 1917, *Indian Jl. Med. Res.*, Vol. 5, p. 350 and Hookworm Infection, 1932, p. 253] In all these privies the practical sanitary point in view is the prevention by the effluent of the conveyance to the soil of any but dead and thereby lightened eggs no longer lying in the sediment, for currents in the tank must not disturb the sediment; for that purpose the design of the concrete L.R.S. privy seems to be the best for the family of a country homestead.

Yours, etc.

October 2, 1939. Clayton LANE.

REVIEWS AND NOTICES.

RICHARDS (Audrey I.) [M.A.(Cantab.), Ph.D.(London), etc.]. **Land, Labour and Diet in Northern Rhodesia. An Economic Study of the Bemba Tribe.**—pp. xiv + 415. With 16 plates, 5 figs., 2 maps & 1 chart. Published for the International Institute of African Languages & Cultures. 1939. London: New York: Toronto: Humphrey Milford, Oxford University Press. [30*s*.]

This book deals with every aspect of the diet of the Bemba, from the preparation of the gardens and sowing of the seed or the gathering of the leaves of wild plants to the sharing-out of the cooked meal to the members of the family circle. The apportioning of land to the head of the family and the effect, on the agricultural output in the villages, of prolonged absence of the males at the mines or European plantations are also discussed.

Charts and Tables are given showing how the men and women occupy their time when at home, the kinship composition of a village and the individual intake of flour from day to day in family groups.

The Bemba, better known as the Awemba to those familiar with North-East Rhodesia in its early days, were a warrior tribe with a highly developed political system. The paramount chief exercised a rigid control over his subjects and was entitled to exact free labour from them for his own or for tribal business. In the course of the last half-century or less, the tribe has had to adapt itself to a peaceful agricultural existence. Latterly has come the attraction of work at the mines, at wages which bring many European-made goods within the workers' reach.

The mode of life of the Bemba has, therefore, undergone more radical changes than has that of most African tribes and they hardly seem to have completely adapted themselves to the changed conditions. As agriculturists, for instance, they are distinctly backward. For this reason it is questionable whether a wise choice was made in the Bemba when seeking a tribe to be the subject of a dietetic study.

In a Foreword to the volume, Dr. Richards modestly states, as her aim, the giving of a broad idea of the extent and variety of nutritional

problems in one African society. She has certainly succeeded admirably in her aim. Moreover, in doing so, she has given a most intimate picture of village life which cannot fail to interest anyone whose lot lies in tropical Africa.

The study of the state of nutrition of African tribes, such as the Bemba, bristles with difficulties which Dr. Richards clearly brings out. With slight modifications, her findings apply to other tribes for, in all such cases, we are dealing with people whose diet is subject to the widest monthly and even daily variations. As Dr. Richards says the "most pronounced feature of this dietary is its alternation between hunger and plenty." A visit from a herd of elephants may convert a well-fed village into a semi-starving one overnight. The early months of the year are always a time of hunger, but drought or locusts may cause a whole year of scarcity. Nothing short of a prolonged study can, therefore, give a true picture of the diet of the native of tropical Africa. The book will be of special interest to the medical man working in East or Central Africa for the light it throws on the many customs and beliefs which govern the growing, preparation and sharing-out of food in the family group. Similar influences, in varying form, are at work in most tribes and the reader of Dr. Richards's book will be stimulated to explore the modifications in his own district and trace their origin. The author has, evidently, a very happy way of winning the confidence of natives and thus gaining an insight into their beliefs and thoughts, such as few Europeans attain even after many years of residence amongst them. The results of her investigations are set out in very readable form. *J. B. Davey.*

Jordan (H.) [A.R.San.I., Instructor of Hygiene, Uganda Medical Service]. **Tropical Hygiene and Sanitation. A Course of Study and Reference Book for Sanitary Inspectors in the Tropics. With Notes on Communicable Diseases and their Prevention** by J. P. Mitchell, M.D., Medical Superintendent & Principal of the Uganda Medical School, **and Medical Entomology** by G. H. E. Hopkins, M.A. (Cantab.), Senior Entomologist, Uganda Medical Department. Baillière's Medical Manuals for Africans.—pp. xiv + 388. With 99 figs. London: Baillière, Tindall & Cox, 7 & 8 Henrietta Street, Covent Garden, W.C.2. [5*s*.]

This volume is number four of a series of Medical Manuals which have been written for the instruction of African nurses, dispensers and health orderlies. The first volume dealt with Anatomy and Physiology and causes of disease, the second with Nursing, and the third with Diagnosis and treatment of disease in the tropics. The present volume is the work of three authors; it aims at embodying in a single text book at reasonable cost all the subject matter, with the exception of anatomy, physiology and law, laid down in the syllabus of studies set out in Appendix II.

Mr. Jordan contributes the first six chapters, which are concerned with matters of general hygiene, such as air and ventilation, water, sanitary building construction, refuse disposal and house drainage, food and meat, disinfection and disinfestation.

Dr. Mitchell contributes chapter seven on communicable diseases; chapter eight, which is the last chapter, is on medical entomology.

Mr. Hopkins, in the entomological section, is careful to point out that the notes which he provides are not of universal application for

Africa. This restriction has enabled him to concentrate on providing the student with characters which are serviceable in distinguishing the important local arthropod vectors of disease.

There are two appendices, of which one gives mensuration notes, while the other deals with the syllabus of the final examination for African sanitary inspectors under the Joint East African Examining Board of the Royal Sanitary Institute.

Reference may be made to a few minor points: no mention is made of chloramine and its advantages over chlorine in the treatment of drinking water; Cyanogas is recommended as best for killing rats and fleas, but in experiments at the Haffkine Institute (1932–35) Calcid briquettes proved much more efficient, 1 gm. of Calcid killing all rats and fleas in the experimental burrows, whereas 42 gm. of Cyanogas dust were required to produce the same effect; in some parts of Malaya long bamboos with the septa cut out somewhat in the manner figured on page 77 collapsed under pressure, while short lengths cut from between adjacent septa proved stronger; *Sarcoptes scabiei*, referred to in chapter 7 as *Acarus scabei*, belongs to the Class Arachnida and not to the Class Insecta.

This manual can be recommended to students and many other readers with every confidence. The subject matter selected is treated in a practical manner and with great conciseness. The illustrations and examples, which have the advantages of being fully explanatory and numerous, will prove most helpful to the student; the lucidity of description is quite unusual. *D. B. Blacklock.*

Rao (V. Venkat) [M.R.San.I., (London), Senior Malaria Inspector, B.N. Railway] & Ramakrishna (V.). [B.Sc. (Hons.), A.R.San.I., Assistant Malaria Inspector, B.N. Railway]. **A Text Book of Malaria Control.**—pp. x+149. 1939. Waltair, R. S. [*Re.* 1.]

This is a book, written by two experienced Malaria Inspectors, designed for the use of Sanitary Inspectors in India who desire to specialize in the malaria side of their work. Its scope is wide. It describes the malaria parasite, the mosquito and its life cycle, identification of anophelines, the epidemiology of malaria, malaria surveys, and nearly the whole field of malaria control. For reasons of economy no illustrations are included, a somewhat serious omission. How difficult it is to write an account of the life cycle of the malaria parasite, or of the morphology of the mosquito larva, for example, that will be intelligible to the uninitiated, for whom, the Foreword states, the book is largely designed, without the generous use of illustrations, is made abundantly clear by the early chapters of the book. Probably it is impossible. A very considerable knowledge of zoological nomenclature is also necessary for the complete understanding of these early chapters.

Perhaps the most helpful chapters of the book are those dealing with practical anti-malaria measures.

More careful editing would increase the value of the book; misspellings are numerous and ambiguity is not always avoided. Such a sentence as "The pupa is comma shaped, the comma forming the abdomen" (p. 15) is not very illuminating.

In short, a plentiful supply of illustrations and some editing might transform the book into a manual for which there appears to be a real need. *Norman White.*

Jack (Rupert W.) [Chief Entomologist]. **Studies in the Physiology and Behaviour of** *Glossina morsitans*, **Westw.**—*Southern Rhodesia. Memoirs of the Department of Agriculture. No. 1.* 1939. May. pp. 203+vii. With 27 figs. & 3 plates. [37 refs.] Salisbury: Govt. Stationery Office.

The first serious account of laboratory work on the physiology of the tsetse fly was published by Buxton and Lewis in 1934 [this *Bulletin*, 1935, Vol. 32, p. 369]. Since then a good deal of similar work has been done, and considerable progress has been made, but the paper under consideration is the first detailed account of experiments in the laboratory, using *Glossina morsitans*. It describes a large number of experiments made, mainly in Salisbury, by Mr. R. W. Jack and his technical assistants.

The results are given in two main sections, the first dealing primarily with physiology and the second with behaviour. The reactions of the adult and pupa were determined under a variety of conditions of temperature and humidity. The results are generally similar to those obtained using other species of tsetse. The insect is seen to be adversely affected by extremes of heat, cold and dryness. On certain processes, *i.e.*, fat metabolism, the author tries to assess the effects of the unnatural conditions inseparable from captivity. The experiments on the behaviour of the flies and their reactions to light and other stimuli are of interest, though it is not certain whether they always indicate the reactions of the wild fly in the field.

The present reviewer considers that all these laboratory experiments reveal something of the physiology of the insect concerned, provided that caution is exercised in the interpretation of the results. Only by laboratory work can we hope to understand fully the physiology of the tsetse, and until the physiology is really understood, satisfactory and permanent control is unlikely. Unfortunately the kind of information obtained in the laboratory is not always of direct practical value, and it is even possible that with misinterpretation it may in practice do more harm than good if the results are applied indiscriminately in the field. Laboratory workers are aware of this and are afraid that their methods may sometimes come into disrepute if the results are put into practice too soon. The information contained in the paper under consideration will serve as a basis for further exact knowledge of *Glossina morsitans*, even if the interpretations the author gives may sometimes have to be modified.

Most tsetse workers seem to have some difficulty in fixing on suitable units for expressing their data. In this paper the normal metric units (including the Centigrade thermometer) are used as a rule, but occasionally the author breaks out into millibars and Fahrenheit, and on one occasion he gives the saturation deficiency of the air in " grams per cubic meter," a unit the reviewer has never found before even in the tsetse literature. Strict uniformity in these matters is surely desirable. *Kenneth Mellanby.*

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Am. signifies Amoebiasis and Amoebic Dysentery.
Bb. ,, Beriberi.
Bl. ,, Blackwater.
B.R. ,, Book Review.
Chl. ,, Cholera.
Der. ,, Tropical Dermatology.
Diet. ,, Deficiency Diseases including Epidemic Dropsy.
Dys. ,, Dysentery (Bacillary and Unclassed).
Fev. ,, Fevers.
Hel. ,, Helminthiasis.
Leish. ,, Leishmaniasis.
Lep. ,, Leprosy.
Lept. signifies Leptospirosis.
Mal. ,, Malaria.
Misc. ,, Miscellaneous.
Oph. ,, Tropical Ophthalmology.
Pel. ,, Pellagra.
Pl. ,, Plague.
Rab. ,, Rabies.
R.B.F. ,, Rat-Bite Fever.
Rep. ,, Medical and Sanitary Reports.
R.F. ,, Relapsing Fever and other Spirochaetoses.
Sp. ,, Sprue.
Tryp. ,, Trypanosomiasis.
Vms. ,, Venoms and Antivenenes.
Y.F. ,, Yellow Fever.
Y. & S. ,, Yaws and Syphilis.

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Azzi, A. S. & Del Frade, A., 395 (Mal.)
Azzi, E., with Magliano, 482 (Fev.)

B.

Baars, J. K., 372 (Chl.)
——, with Mochtar, 371 (Chl.)
Baba, H., with Kubo, 504 (Misc.)
——, with —— & Gomi, 504 (Misc.)
Babenuishev, V. P. & others, 972 (Pl.)
Bablet, J., 29 (Y.F.)
——, with Bertrand & Block, 1019 (Lep.)
Baccialone, L., with Farinaud, Lataste & Canet, 816 (Mal.)
Bachman, G. W., 147 (Hel.)
Bacigalupo, J., 600, 605, 606 (Dys.), 626 (Hel.)
—— & Aguirre Pequeno, E., 251 (Hel.)
Badenski, G. & Bruckner, I., 193 *bis* (Rab.)
——, with Ciuca, Ionescu & Teriteanu, 823 (Mal.)
——, with Shute, 393 (Mal.)
——, with Tupa, Ciuca & Ionescu, 822 (Mal.)
Bahr, L., 978 (Pl.)
Bailly, J., with Remlinger, 194, 195, 196, 200 *bis*, 726 *bis*, 727, 731, 732 (Rab.)
Bajolet, 947 (Misc.)
Baker, F. C., with Bennett & Sellards, 639 *bis* (Y.F.)
Baliña, P. L. & Basombrio, G., 238 (Lep.)
Ballif, L., with Ciuca, Chelaresco & Lavrinenko, 11, 390, 924 (Mal.)
Balozet, L., 199, 730 (Rab.)
Baltazard, M., with Blanc, 471 (Fev.)
——, with —— & Donnadieu, 464 (Fev.)
Balteanu, I., with Ciuca, Alexa, Boeriu, Rugina, Stretcu & Radianov, 11 (Mal.)
——, with ——, Francke, Alexa & Micu, 11 (Mal.)
——, with Slatineanu, Nitulescu & Levit, 545 (Pel.)
——, with ——, Sibi, Nitzulesco & Levit, 545 (Pel.)
Balteanu, J., with Ciuca, Alexa, Balteanu, R., Boeriu & Radianov, 11 (Mal.)
Balteanu, R., with ——, Balteanu, J., Alexa, Boeriu & Radianov, 11 (Mal.)
Banerjea, J. C. & Bhattacharya, P. B., 699 (B.R.)
Banerjee, D. N., (365), 378, 381, 901 (Chl.)
Barbosa, A., 12 (Mal.)
Bardswell, N. D., 342, 343, 869 (Misc.)
Baré, J., 1017 (Lep.)
——, with Kervingant, 1016 (Lep.)
Barker, W. H. & Rhoads, C. P., 57 (Sp.)
Barlow, C. H., with Scott, 36 (Hel.)
Barnett, E. J., 575 (Vms.)
Barnett, L., 251 (Hel.)
Barreto, J. de B., 977 (Pl.)
Barros, E. F., 574, (866) (Vms.)
Barthas, E., 31 (Y.F.)
Bartholomeusz, F. E. R., 540 (Lep.)
Basnuevo, J. G. & Anido, V., 35, 320 (Hel.)
——, with Fonseca & Kouri, 593 (Hel.)
——, with Kourí, Sotolongo & Anido, 599, 605 (Dys.), 626 (Hel.)
Basombrio, G., with Balina, 238 (Lep.)
Basu, B. C., 587 (Mal.)
——, with Chopra, 786 (Mal.)
Basu, N. K., 246 (Lep.)
Basutoland, 234 *bis* (Lep.)
Bates, M., (780) (Mal.)
Bauersfeld, E. H., 998 (Fev.)
Baumann, H., with Schwetz, 777 (Mal.)
Beach, W. R., 199 (Rab.)
Bean, H., with Mackenzie, 599 (Dys.)
Bean, W. B., with Spies, Aring & Gelperin, 553 (Pel.)
——, with —— & Stone, 554 (Pel.)
——, with Vilter & Spies, 554 (Pel.)
Bearup, A. J. & Morgan, E. L., 721 (Hel.)
Bechell, L. M. (535) (Lep.)
Becker, F. E., with d'Amour & van Riper, 571 (Vms.)
Beeuwkes, H., 899 (Chl.)
Benchetrit, A., (697) (Lep.)
di Benedetto, V., 766 (R.F.)
Bennett, B. L., Baker, F. C. & Sellards, A. W., 639 *bis* (Y.F.)
Benzoni, G., 289 (Am.)
Bequaert, J. C., with Shattuck, Sandground, Hilferty & Clark, 607 (B.R.)
Berberian, D. A., 1034, 1035 (Leish.)
Bergier, P., with Giraud, 1026 *bis* (Leish.)
——, with —— & Cabassu, 1026 (Leish.)
Berlin, C., 946 (Misc.)
Bernal Londoño, M., with Morales Muñoz & de Souza Araújo, 690 (Lep.)
Bernard, M., with Pigoury, 985 (Fev.)
Bernard, P. N., Guillerm, J. & Gallut, J., 373 *bis*, 374 (Chl.)
Bernardino, S., with Falla Alvarez & Silveira, 605 (Dys.)

Bernier, G. L., with Laurens & Fort, 1000 (Fev.)
Bernkopf, H., with Kligler, 193, 724, 728 (Rab.)
Bernth, O. & Stürup, G. K., 552 (Pel.)
Berry, G. P., with Morrow, Syverton & Stiles, 772 (Lept.)
Berté, M., 671 (Tryp.)
Bertram, D. S., 506 (Misc.)
Bertrand, I., Bablet, J. & Block, F., 1019 (Lep.)
Bessemans, A., Wittebolle, P. & de Borchgrave, O., 769 (Lept.)
Betz, H., 247 (Lep.)
van Beukering, J. A., 834 (Hel.)
Bevere, L., 490 (Mal.)
——, with van Thiel, 778 (Mal.)
——, with ——, Reuter & Sautet, 1004 (Mal.)
Beveridge, A. J., 419 (Misc.)
Bhaduri, B. N., 957 (Oph.)
Bhalerao, G. D., (37) (Hel.)
Bhargava, R. C., 971 (Pl.)
Bharucha, K. H., with Wats, 10 (Mal.)
Bhattacharya, D. P., with Ghosh & De, 855 (Vms.)
Bhattacharya, P. B., with Banerjea, 699 (B.R.)
Bibb, J., with Diggs, 936 (Misc.)
Bickel, G., 543 (Pel.)
Bing, J. & Broager, B., 62 (Sp.)
Binns, H. R., 747 (Tryp.)
Biozzi, S., 223 (Tryp.)
Birger, C., with Dubois & Vitale, 842 (Hel.)
Bishop, A., Tate, P. & Thorpe, M. V., 587 (Mal.)
Bishopp, F. C. & Smith, C. N., 475 (Fev.)
Bispham, W. N., 500 (Mal.)
Bisquerra, E. D., 873 (B.R.)
Bistrenin, A., 978 (Pl.)
Blacklock, D. B., 336 (Mal.)
Blair, D. M., 133 (Mal.), 736 (Tryp.)
Blanc, G., 1028 (Leish.)
—— & Baltazard, M., 471 (Fev.)
—— & ——, with Donnadieu, A., 464 (Fev.)
—— & Noury M., 982 (Fev.)
Blatt, M. L., Hoffman, S. J. & Schneider, M., 196 (Rab.)
Blewitt, B., 461 (Fev.)
Block, F., with Bertrand & Bablet, 1019 (Lep.)
Bloom, W. & Taliaferro, W. H., 586 (Mal.)
Bobkoff, G., with Gavrilov & Laurencin, 929 (Mal.)
Bock, E., 924 (Mal.)
——, with Weyer, 920 (Mal.)
Boecker, E. & Jahn, G., 729, (Rab.)
Boedyn, K. B. & Verbunt, J. A., 103 (Der.)
Boenjamin, R., 244, 245 (Lep.)
de Boer, H. S., 429 (Misc.)
Boeriu, V., with Ciuca, Balteanu, Alexa, Rugina, Stretcu & Radianov, 11 (Mal.)
——, with ——, Balteanu, J., Alexa, Balteanu, R., & Radianov, 11 (Mal.)
Boers, E. R. J., Kouwenaar, W. & Wolff, J. W., 102 (Der.)
Bogart, C. N., 553 (Pel.)
Boggino, J., with Gatti & Prieto, 1032 (Leish.)
Bohlander, L., with Walch-Sorgdrager & Schüffner, 116 (Lept.)
Boisseau, R., 466 *bis*, 473 (Fev.)
Boldt, E., with Sioli & Kentenich, 494 (Mal.)
Boletin Sanitario, Buenos Aires, 228 (Tryp.) 962 *bis* (Pl.)
Bombay, 729 (Rab.), 976, 977 (Pl.), 1008 (Mal.)
Boné, G., 759 *bis*, 760 *bis* (R.F.)
Bonne, C., 838 (Hel.)
Bonnefoi, A., with Mollaret, 774 (R.B.F.)
Bonnet, R., with Vigne, Vignoli & Tivollier, 693 (Lep.)
Bonnin, H. & Aretas, R., 304 (Am.)
von Bonsdorff, B., (250) (Hel.)
Bonsignore, A., 294 (Am.)
Boquet, P., 860 *bis*, 862 (Vms.)
——, with Césari, 860 (Vms.)
de Borchgrove, O., with Bessemans & Wittebolle, 769 (Lept.)
v. Bormann, F., 853 (Vms.)
Boscq, H., with Guibert, 216 (Tryp.)
Bose, R., with Greval & Lowe, 537 (Lep.)
Bose, S., 895, 901 (Chl.)
——, with Read, Singh & Seal, 895 (Chl.)
Boselli, A., with Vierthaler, 742 (Tryp.)
Boswijk, J. C., with de Langen & van Nieuwenhuizen, 545 (Pel.)
Botreau-Roussel, with Assali, Dejou, Huard, Montagné, Pales & Pales & Roques, 431 (B.R.)
Bottema, C. W., 44 (Y. & S.)
Botzaris, A., 470 *bis* (Fev.)
Bouček, J., 729 (Rab.)
Boudouresque, with Pieri & Sardou, 295 (Am.)
Boudouresques, J., with Roger, 784 (Mal.)
——, with —— & Lombard, (535) (Lep.)
Boudymko, F. A., with Danilova, 491 (Mal.)
Boughton, D. C. & Byrd, E. E., 598 (Dys.)
Bourret, R., 563 (Vms.)
Boursnell, J. C., Dangerfield, W. G. & Wormall, A., 668 (Tryp.)
Boyd, G. H., 1011 (Mal.)
Boyd, L. J. & Schlackmann, M., 10 (Mal.)
Boyd, M. F., 495, 808 (Mal.)
—— & Kitchen, S. F., 492, 495, 497, 682 *bis*, 811 (Mal.)
——, —— & Matthews, C. B., 922 (Mal.)
——, Kupper, W. H. & Matthews, C. B., 493 (Mal.)
—— & Matthews, C. B., 780, 809 (Mal.)
Bozicevich, J., (146), 847 (Hel.)
———, with Wright & Gordon, 846 (Hel.)
Bradfield, E. W. C., 343 (Misc.)
Brady, F. J., with Wright, 329 (Hel.)
v. Brand, T. & Otto, G. F., 325 (Hel.)
de Brauwere, P., 655 (Tryp.), 697 (Lep.)
Brazzaville : Afrique Equatoriale Française, 632 (Y.F.), 658 (Tryp.).
Breininger, D., 964 (Pl.)
Breton, M. & Lavier, G., 718 (Hel.)
Breuer, A., 603 (Dys.)
Breuil & Mollaret, 331 (Mal.)
van Breuseghem, R., 536 (Lep.)
Briercliffe, R., 736 (Tryp.)
Briese, R. R. & Couch, J. F., 411 (Misc.)
Brigham, G. D., 477 *bis* (Fev.)
Brink, C. J. H. & Das Chowdhury, D. K., 1009 (Mal.)
Brissolese, A., 141 (Mal.)

British Empire Leprosy Relief Association, 1015 (Lep.)
British Medical Journal, 552 (Pel.), 942 (Misc.)
Broager, B., with Bing, 62 (Sp.)
Broderick, M. J. & Pijper, A., 659 (Tryp.)
Bromfield, R. J., with Fairley, 120, 885 (Bl.)
——, with ——, Foy & Kondi, 422 (Misc.)
Broom, J. C., 662 (Tryp.)
—— & Brown, H. C., 660 (Tryp.)
——, with ——, 217 (Tryp.)
——, with Hoare, 661 (Tryp.)
Brown, A. A. F., 223 (Tryp.)
Brown, H. C., 218 (Tryp.)
—— & Broom, J. C., 217 (Tryp.)
——, with ——, 660 (Tryp.)
Brown, M. H. S., with Young, 949 (Misc.)
Brown, P. W. & Hodgson, C. H., 299 (Am.)
Bruckner, I., with Badenski, 193 *bis* (Rab.)
Brumpt, E., 404, 928 (Mal.), 753 (Tryp.)
Brumpt, L., with Pinard & Raymondeau, 667 (Tryp.)
Bruneau & Chapuis, 999 (Fev.)
Bruneau, J., 992, 993 (Fev.)
——, with Genevray, 378 (Chl.)
——, with —— & Seyberlich, 904 (Chl.)
Brussin, A. M. & Sternberg, E. J., 112, 113 (R.F.)
Btesh, S., with Shapiro, 1031 (Leish.)
de Buck, A., 489 (Mal.)
——, with Swellengrebel, 90 (B.R.)
Buckley, J. J. C., 40, 627 (Hel.)
Budac, O., with Pop, 743 (Tryp.)
Bühler, F. & v. Hasselbach, H., 715 (Hel.)
Bulletin of the Health Organisation (League of Nations), 11 (Mal.)
Bulletin de l'Institut d'Hygiène du Maroc, 686 (Mal.), 988 (Fev.)
Bulletin de l'Office International d'Hygiène Publique, 19, 28, 648 (Y.F.), 780 (Mal.)
Bulletin of the Ophthalmological Society of Egypt, 957 (Oph.)
Bürgi, K., 626 (Hel.)
Burnet, E., 240, 538, 539, 698 (Lep.)
——, Cuénod, E. & Nataf, R. 955 (Oph.)
——, E., Jadfard, H. & Larabi, M., 105 (Der.)
Burschkies, K., (697) (Lep.)
Busch, H.-G., 388 (Mal.)
Buşila, L., with Mezincesco, Cornelson & Lazar, 11 (Mal.)
Buttle, G. A. H., 941 (Misc.)
Buxton, P. A., 308 (Pl.)
Buxton, R., 957 (Oph.)
Bynum, W. T., 943 (Misc.)
Byrd, E. E., with Boughton, 598 (Dys.)

C.

Caballero, E., with Nieto Roaro, 624 (Hel.)
Cabassu, H., with Giraud & Bergier, 1026 (Leish.)
Caiado, I. de C., 723 (Hel.)
Calcutta, (905) (Chl.)
Calcutta: All-India Institute of Hygiene & Public Health, 449 (Leish.), 889 (Bl.)
Calcutta Medical Journal, 907 (Diet.)
Callender, G. R. & Gentzkow, C. J., 679 (Mal.)
——, with Gentzkow, 257 (Mal.)
——, with Simmons, Curry, Schwartz & Randall, 874 (B.R.)
Callot, J., 638 (Y.F.)
—— & Ristorcelli, A., 136 (Mal.)
Cambessédès, H., 30 (Y.F.)
Cambournac, F. J. C., 920 (Mal.)
Campbell, H. E., 40 (Hel.)
van Campenhout, J., 19 *bis* (Y.F.), 966 (Pl.)
Canellakis, A. P., with Livadas & Valaoras, 397 (Mal.)
Canet, J., 818 (Mal.)
——, with Farinaud, Lataste & Baccialone, 817 (Mal.)
Cappelli, E., 538 (Lep.)
Cardoso, F. A., 228 (Tryp.)
Carlinfanti, E., 771 (Lept.)
Carmichael, J., 346 (Misc.)
Carnahan, C. T., 640 (Y.F.)
Caro, 383 (Chl.)
Carol, W. L. L. & Prakken, J. R., (51) (Y. & S.)
Carpano, M., 214 (Tryp.)
Carrillo, D. F., 228 (Tryp.)
Carrión, A. L. & Pimentel-Imbert, M. F., 99 (Der.)
Carroll, T. B., with Robinow, 577 (Vms.)
Carrot & Fabiani, G., 392 (Mal.)
Cassiano, T. P., 535 (Lep.)
Castellani, A., (62) (Sp.), 105 (Der.), 963 (Pl.)
—— & Acanfora, G., 254 (Hel.)
—— & Amalfitano, G., 454 (Leish.)
Castellani, G. T., 567 (Vms.)
Castellano, T. & Garzon, R., (558) (Pel.)
Castellanos, A., (597) Dys.
Castillo Odena, I., 325 (Hel.)
Catanei, A., 104 (Der.)
——, with Parrot, 388 (Mal.)
——, with ——, Ambialet & Clastrier, (688), 812 (Mal.)
Causey, O. R., 505 (Misc.)
Cawston, F. G., 38 *bis*, (39), 619, 620 (Hel.), 454 (Leish.)
Ceccaldi, J., with Saleun & Palinacci, 32 (Y.F.)
Césari, E. & Boquet, P., 860 (Vms.)
Chabeuf, M., 153 (Hel.)
Chagas, A. W., 449 (Leish.)
——, with Ferreira, Mangabeira & Deane, L., 449 (Leish.)
Chagas, E., da Cunha, A. M., Ferreira, L. C., Deane, L., Deane, G., Guimarães, F. N., von Paumgartten, M. J. & Sá, B., 446 (Leish.)
Chambers, H. D., 45 (Y. & S.), 88 (B.R.)
Chanco, P. P., Jr., 723 (Hel.)
Chandra, S. N., with Greval & Woodhead, 935 (Misc.)
——, with Roy & Siddons, 681 (Mal.)
Chang, F. C., with Chung, 763 (R.F.)
Chang, S., 107 (R.F.)
Chang, S. P., 955 (Oph.)
Chapuis, with Bruneau, 999 (Fev.)
Charamis, J. S., with Cosmetatos, 456 (Oph.)
Charbonnier, 501 (Mal.)
Chatellier, L., 106 (Der.)
Chatterjee, B. C., (379) (Chl.)
——, with Pasricha & DeMonte, 379 (Chl.)
——, with ——, —— & Mian, 903 (Chl.)

Chatterjee, D. N. & Malik, K. S., 379 (Chl.)
——, with Pasricha & Das, 377 (Chl.)
——, with —— & Paul, 383, 902 (Chl.)
Chatterjee, H., 902 (Chl.)
Chatterjee, M. L., (247) (Lep.)
Chatterjee, S. K. & Deo, L. R. S., 381 (Chl.)
Chatterji, S. N., with Lowe, 534, 1015 (Lep.)
Chaudhuri, R. N., with Sen Gupta, Rai Chaudhuri & Napier, 543 (Pel.)
Chaudhuri, S. & Mangalik, V., 424 (Misc.)
Checcacci, L., 897 (Chl.)
Chédécal, 779, 1006 (Mal.)
Chelaresco, M., with Ciuca, Ballif & Lavrinenko, 11, 390, 924 (Mal.)
Chenderowitch, R., with Fastovskaia, 683 (Mal.)
Chessa, F., 813 (Mal.)
Chin, T., with Winfield, 319 (Hel.)
Chinese Medical Journal, 331 (Mal.).
Chisar, with Litarczek, (717) (Hel.)
Chodzko, W., (601) (Dys.)
Chopra, R. N. (260), 396 (Mal.), (905) (Chl.)
—— & Basu, B. C., 786 (Mal.)
——, with Chowhan, 695 (Lep.)
—— & Das Gupta, B. M., 1012 (Mal.)
——, with ——, 583 (Mal.)
——, —— & Sen, B., 499 (Mal.)
——, Pasricha, C. L., Goyal, R. K., Lal, S. & Sen, A. K., 909 (Diet.)
——, with Roy, 566 (Vms.)
Choremis, K. & Spiliopoulos, G., 398 (Mal.)
Chorine, V., 340, 394 (*bis*), (Mal.)
——, with Marchoux, 241, 540, 698 (Lep.)
Chortis, P., 930 *bis* (Mal.)
Chow, C. Y., (834) (Hel.)
——, with Hsü, 144 (Hel.)
Chowhan, J. S. & Chopra, R. N., 695 (Lep.)
Christophers, R. & Sinton, J. A., 390 (Mal.)
—— & Fulton, J. D., 340 (Mal.)
Chu, H. J., (41), 625 (Hel.)
——, with Hoeppli & Feng, 35 (Hel.)
Ch'u, Y.-C. & Forkner, C. E., 417 (Misc.)
Chue Sufui, with Schratzenmayr & Tsen Hyen-Wun, 445 (Leish.)
Chung, H., 450 (Leish.)
Chung, H.-L. & Chang, F. C., 763 (R.F.)
——, with Feng, 110 (R. F.), 1025 (Leish.)
—— & ——, 111 (R.F.), 1025 (Leish.)
——, with —— & Hoeppli, 1027 (Leish.)
——, with Ts'un, 111 (R.F.)
—— & T'ung, T., 715 (Hel.)
—— & Wei, Y.-L., 763 (R.F.)
Cicchitto, A. M., 392, 678 (Mal.), 601 (Dys.)
Cicchitto, E., 37 (Hel.)
Ciferri, R. & Redaelli, P., 601 (Dys.)
Cilento, R., 1017 (Lep.)
Ciuca, M., 11 (Mal.)
——, Badenski, G., Ionescu, P. & Teriteanu E., 823 (Mal.)
——, Ballif, L., Chelaresco, M. & Lavrinenko, M., 11, 390, 924 (Mal.)
——, —— & Alexa, with Balteanu, R., Boeriu, V. & Radianov, A., 11 (Mal.)
——, Balteanu, I., Alexa, I., with Boeriu, V., Rugina, I., Stretcu, I. & Radianov, A., 11 (Mal.)
——, ——, Francke, M., Alexa, E. & Micu, I., 11 (Mal.)
Ciuca, M. & Tupa, with Badenski, G. & Ionescu, 822 (Mal.)
Clark, H. C., (926) (Mal.)
—— & Komp, W. H. W., 819 (Mal.)
Clark, S. D., with Shattuck, Bequaert, Sandground & Hilferty, 607 (B.R.)
Clastrier, J., with Parrot, Catanei & Ambialet, (688), 812 (Mal.)
Claudian, I. & Ghermani, A., 546 (Pel.)
Cleave, T. L., 990 (Fev.)
Cleland, J. B., 108 (R.F.)
Clement, L., 592 (Hel.)
Clemente, G., 943 (Misc.)
Clerk, M., 24 (Y.F.)
Clerckx, 696 (Lep.)
Co, C., with Yumoto, 36 (Hel.)
Cochrane, E., 343 (Misc.)
Cochrane, R. G. & Rajagopalan, G., 238 (Lep.)
Coelho, J. T., 244 (Lep.)
Coggeshall, L. T., 824 (Mal.)
—— & Eaton, M. D., 404, 405 (Mal.)
——, with ——, 926 (Mal.)
—— & Kumm, H. W., 406 (Mal.)
——, with Milam, 583 (Mal.)
Colarusso, A., 773 (R.B.F.)
Colas-Belcour, J., with Roubaud & Mathis, 928 (Mal.)
——, with —— & Stefanopoulo, 638 (Y.F.)
Collignon, E., 136, 333, (680) (Mal.)
Colonial Development Fund (Malarial Research Scheme), 126, 387 (Mal.)
Colonial Office, 409 (Misc.)
Colson, M., with Jacotot & Le Roux, 196 (Rab.)
Condell, L. A., (943) (Misc.)
Congo Belge, 408 (Misc.), 656 (Tryp.)
Connell, J. H., 726 (Rab.)
Constantinescou, P., with Grecou & Ionescou, 552 (Pel.)
——, with Ionesco, 548 (Pel.)
Contreras, M., with Macchiavello, (315) (Pl.)
Cook, D., 409 (Misc.)
Cooley, R. A., (999) (Fev.)
Cooper, M., 343 (Misc.)
Corica, P., with De la Barrera, (316) (Pl.)
Corkill, N. L., 502 (Misc.)
Cornelson, D. A., with Mezincesco, Lazar & Buşila, 11 (Mal.)
Cornet, E., 250 (Hel.), 456 (Oph.)
Corradetti, 677 (Mal.)
Corradetti, A., 1005 (Mal.)
Corson, J. F., 208 *bis*, 210 *bis*, 662, 667, 737, 741 (Tryp.)
Cosmetatos, G. F. & Charamis, J. S., 456 (Oph.)
Couch, J. F., with Briese, 411 (Misc.)
Courtney, K. O., 1016 (Lep.)
Coutelen, F., 252, 715 (Hel.)
Cova-Garcia, P., (780) (Mal.)
Covell, G., 917 (Mal.)
Cowdry, E. V., 538 (Lep.)
—— & Ravold, A., 241 (Lep.)
Cox, H. R., 479, 984 (Fev.)
——, with Davis, 479 (Fev.)
Crabtree, H. S., with Mackie, 32 (Y.F.)
Craig, C. F. & Swartzwelder, J. C., 294 (Am.)
Cram, E. B. & Reardon, L., 845 (Hel.)
Crawford, R., (811) (Mal.)

Cremer, S. de G., 690 (Lep.)
Cristiani, M., 661 *bis* (Tryp.)
Crocker, C. G., with Pijper, 468 (Fev.)
Crosby, B. L., with Schapiro & Sickler, 146 (Hel.)
Crouch, H. A., 18 (Y.F.)
Cruveilhier, L., Dieryck, J. & Viala, C., 732 (Rab.)
——, Lépine, P. & Viala, C., 197 (Rab.)
——, with Nicolau, Truche, Kopciowska & Viala, 195 (Rab.)
—— & Viala, C., 197 (Rab.)
Cuénod, E., with Burnet & Nataf, 955 (Oph.)
Culbertson, J. T., 590 (Hel.), 751 *ter*, 752 (Tryp.)
—— & Kaplan, S. S., 148 (Hel.)
—— & Kessler, W. R., 750 (Tryp.)
—— & Kolodny, M. H., 231 (Tryp.)
——, with Zwemer, 748 (Tryp.)
Cumming, H. S., 647 (Y.F.)
Cummins, S. L., 343 (Misc.)
da Cunha, A. M., 447 *ter*, 1027 (Leish.)
——, with Chagas, Ferreira, Deane, L., Deane, G., Guimarães, von Paumgartten & Sá, 446 (Leish.)
—— & Dias, E., 451, 1035 (Leish.)
Curasson, G., 414 (Misc.)
Currie, J. G., with Sorley, 400 (Mal.)
Curry, D. P., with Simmons, Callender, Schwartz & Randall, 874 (B.R.)

D.

van Daele, G., 913 (Diet.)
Daengsvang, S. & Tansurât, P., 156 (Hel.)
Dangerfield, W. G., with Boursnell & Wormall, 668 (Tryp.)
Danglemont, F., with Jolly, 782 (Mal.)
Daniel, with d'Oelsnitz, Saurin & Raibaudi, 451 (Leish.)
Danilova, M. I. & Boudymko, F. A., 491, (Mal.)
Dao Van Thai, with Hérivaux & Roncin, 918 (Mal.)
Das, B. K., with White, 14 (Mal.)
Das, P. C., with Lahiri, 377 (Chl.)
——, with Pasricha & Chatterjee, 377 (Chl.)
——, with —— & Lahiri, 367 (Chl.)
Das Chowdhury, D. K., with Brink, 1009 (Mal.)
Das Gupta, B. M., 114 (Lept.), 1006 (Mal.)
——, with Chopra, 1012 (Mal.)
—— & ——, 583 (Mal.)
——, with —— & Sen, 499 (Mal.)
Das Gupta, N. C., 1033 (Leish.)
Dassanayake, W. L. P., 834 (Hel.)
Dastidar, S. K. G., 1031 (Leish.)
Datta Roy, B. K., with Vardon, 377 (Chl.)
Davey, T. H., (469) (Fev.)
—— & Gordon, R. M., 582 (Mal.)
David, J., 622 (Hel.)
Davies, H. N., 343, 344 (Misc.)
Davis, A. P., 1017 (Lep.)
Davis, E. C., 300 (Am.)
Davis, G. E. & Cox, H. R., 479 (Fev.)
—— & Parker, R. R., 477 (Fev.)
——, with ——, 479 (Fev.)
Davy, J. B., 665 (Tryp.)
Day, P. L., with Shukers & Langston, 424 (Misc.)
De, S. S., with Ghosh, 856 (Vms.)
——, with —— & Bhattacharya, 855 (Vms.)
Deane, G., with Chagas, da Cunha, Ferreira, Deane, L., Guimarães, von Paumgartten & Sá, 446 (Leish.)
Deane, L., with Chagas, da Cunha, Ferreira, Deane, G., Guimarães, von Paumgartten & Sá, 446 (Leish.)
——, with Ferreira, 754 (Tryp.)
——, with ——, Mangabeira & Chagas, 449 (Leish.)
—— & Jansen, G., 754 (Tryp.)
Debdas, N., 914 (Diet.)
Debono, J. E., 942 (Misc.)
DeBusk, C. K., 333 (Mal.)
Decourt, P., 498, 782, 818 (Mal.)
——, with Deschiens, 292 (Am.)
—— & Schneider, J., 586 *bis* (Mal.)
Degotte, J., 535, 1016 (Lep.)
——, with Dubois, 535 (Lep.)
Déjou, L., 842 (Hel.), 948 (Misc.)
——, with Advier, 842 (Hel.)
De la Barrera, J. M., with Alfarado, 962 (Pl.)
—— & Corica, P. (316) (Pl.)
Delacoux des Roseaux, with Valleteau & Duliscouet, 450 (Leish.)
Delbove, P., with Alain, 947 (Misc.)
Del Favero, E., 374 (Chl.)
Del Frade, A., with Azzi, 395 (Mal.)
Delidimitriou, G., 747 (Tryp.)
Delpy, L. & Rafyi, A., 757 (R.F.)
Del Vecchio, G., (681) (Mal.)
Démina, P. A., 932 (Mal.)
DeMonte, A. J. H., (905) (Chl.)
—— & Gupta, S. K., 379 (Chl.)
——, with Pasricha & Chatterjee, 379 (Chl.)
Dempster, G. O., 320 (Hel.)
Denéchau, D. & Mandroux, J., 771 (Lept.)
Denecke, K., with van den Berghe, 628 (Hel.)
Denovan, A. E. B., 839 (Hel.)
Deo, L. R. S., with Chatterjee, 381 (Chl.)
Deprecq, M., with Pauly, 546 (Pel.)
Derrick, E. H., (469) (Fev.)
Deschiens, R., 284, 287, 290 *bis*, 291 (Am.), 830 (Hel.)
—— & Decourt, P., 292 (Am.)
——, with Dopter, 285 *bis*, 286 (Am.)
—— & Provost, A., 305 (Am.)
——, with Roubaud, 833 (833) (Hel.)
Des Essarts, J. Q., 540 (Lep.)
Desnos, E. H. & Abély, P., 142 (Mal.)
Destribats, with Sanner, 483 (Fev.)
Devignat, R., 969 *bis* (Pl.)
Devine, J., 13 (Mal.)
Dexter, L., with Gardner, 783 (Mal.)
Diamantis, A., 622 (Hel.)
Dias, E., 230 (Tryp.), 477 (Fev.)
——, with da Cunha, 451, 1035 (Leish.)
——, with Philip, 998 (Fev.)
—— & Torrealba, J. F., 229 (Tryp.)
Díaz, B., 323 (Hel.)
Dickson, E. C., 508 (Misc.)
—— & Gifford, M. A., 507 (Misc.)
Dieryck, J., with Cruveilhier & Viala, 732 (Rab.)
Diggs, L. W. & Bibb, J., 936 (Misc.)
Digonnet, L. & Mathis, M., 765 (R.F.)

Dikshit, with Nandi, 1008 (Mal.)
Dinger, J. E., 23 (Y.F.)
Dobell, C., 597 (Dys.)
Dobriner, K., Strain, W. H. & Localio, S. A., 547 (Pel.)
Dodero, J., 196, 198, 731 (Rab.)
Dominguez, F., (639) (Y.F.)
Donatien, A., with Sergent, E., Parrot & Lestoquard, 1026 (Leish.)
Donnadieu, A., with Blanc & Baltazard, 464 (Fev.)
Donohoe, H. C., 570 (Vms.)
Donovan, A., 650, 651 (Y.F.)
Dopter, C. & Deschiens, R., 285 *bis*, 286 (Am.)
Dos Anjos, W. V., with Martins, 618 (Hel.)
Doubrow, S., 322 (Hel.)
Doull, J. A., 533 (Lep.)
Doval, J. M., with Kourí, 720 (Hel.)
Dozortseva, P. M., 259 (Mal.)
Dubois, A. & Degotte, J., 535 (Lep.)
—— & Forro, M., 842 (Hel.)
——, with Gavrilov & Fester, 698 (Lep.)
——, Vitale, S. & Birger, C., 842 (Hel.)
Duca, C. J., 750 (Tryp.)
Duliscouet, with Valleteau & Delacoux des Roseaux, 450 (Leish.)
Dumas, J., 700 (B.R.)
Dumitresco, D., with Moisesco & Paidiu, 717 (Hel.)
Dupouy, 989 (Fev.)
Durand, P., 814 *bis* (Mal.), 970 *bis* (Pl.)
—— & Villain, 932 (Mal.)
Durand, R., with Laigret, 993 (Fev.)
Duran-Reynals, F., 563, 857 (Vms.)
Duren, A., 780 (Mal.)
Durieux, C., with Peltier, Jonchère & Arquié, 637 (Y.F.)
Dusan, J., Lombard, R. & Tivollier, (535) (Lep.)
Dutton, L. O., 159 (Misc.)
Dyer, R. E., 479, 986, 991 (Fev.)
Dyke, H. W., 234 *bis* (Lep.)

E.

Earle, K. V., 415 (Misc.)
East African Medical Journal, 841 (Hel.)
Eaton, M. D., 405, 821 (Mal.)
—— & Coggeshall, L. T., 926 (Mal.)
——, with ——, 404, 405 (Mal.)
Eddy, H. M., with Myers, 933 (Misc.)
Edwards, W. P. S., 198 (Rab.)
Eggleton, W. G. E., (561) (Bb.)
Ejercito, A., 335 (Mal.)
Ekblom, T., 134 (Mal.)
El Gazayerli, M., 619 (Hel.)
El Gholmy, with Mahdi & Shukry, 912 (Diet.)
——, with Shukry & Mahdi, 912 (Diet.)
Enderle, C., (296) (Am.)
Erhardt, A., 625 (Hel.)
Ermen, J., 1032 (Leish.)
Eschbach, H., 771 (Lept.)
Escomel, E., 428 (Misc.)
Eskey, C. R., 310 (Pl.)
—— & Haas, V. H., 968 (Pl.)
Eskridge, L., with Hegner, 338, 585 (Mal.)
Esnault, P. & Loubet, R., 453 (Leish.)
Essed, W. F. R., with Müller & Hazebroek, F. E. A., 99 (Der.)
Esseveld, H. & Mochtar, A., 115, (770) (Lept.)
Evans, B. D. F. & Wills, L., 936 (Misc.)
Evans, F. L., 1021 (Lep.)
Evans, K. L. & Arnold, L. E., 412 (Misc.)
Evans, R. R., 719 (Hel.)
Evans, T., 492 (Mal.)
Everling, W., 731 (Rab.)

F.

Fabbri, G., 567 (Vms.)
Fabiani, G., with Carrot, 392 (Mal.)
Fabre, M., (697) (Lep.)
Faget, G. H., Palmer, M. R. & Sherwood, R. O., 260 (Mal.)
Faiguenbaum, J. & Gasic, G., 497 (Mal.)
Fairley, N. H., 118, 885, 941 (Bl.), 422 (Misc.)
—— & Bromfield, R. J., 120, 885 (Bl.)
——, ——, Foy, H. & Kondi, A., 422 (Misc.)
Falla Alvarez, L., Silveira, R. & Bernardino, S., 605 (Dys.)
Farinaud, E., Lataste, C., Baccialone, L. & Canet, J., 816 (Mal.)
—— & Ragiot, C., 786 (Mal.)
——, Ramijean, R. & Favot, M., 679 (Mal.)
Farinaud, M., 970 (Pl.)
Farinaud, M. E., 581 (Mal.)
——, with Lataste & Nguyen-Van-Lien, 815 (Mal.)
Fastovskaia, E. & Chenderowitch, R., 683 (Mal.)
Fattovich, G. & Lenti, P., 262 (Mal.)
Faust, E. C., d'Antoni, J. S., Odom, V., Miller, M. J., Peres, C., Sawitz, W., Thomen, L. F., Tobie, J. & Walker, J. H., 144 (Hel.)
Favilli, G., 858 (Vms.)
Favorissova, B., 965 (Pl.)
——, with Korobkova & Krainova, 967 (Pl.)
Favot, M., with Farinaud & Ramijean, 679 (Mal.)
Federated Malay States, 128 (Mal.), 197 (Rab.), 382 (Chl.), 463 (Fev.)
Fedorov, V., with Lobanov, 966 (Pl.)
——, with Shmelev, (964) (Pl.)
Feijóo, E. J. C., with Mazza & Jörg, 227 (Tryp.)
Feissolle, L., with Raynal & Lieou, 896 (Chl.)
Feldberg, W., Holden, H. F. & Kellaway, C. H., 565 (Vms.)
—— & Kellaway, C. H., 564 (Vms.)
Felix, A., 941 (Misc.)
Feng, L.-C. & Chung, H.-L., 110 (R.F.), 1025 (Leish.)
——, with Chung, 111 (R.F.), 1025 (Leish.)
——, —— & Hoeppli, R., 1027 (Leish.)
——, with Hoeppli & Chu, 35 (Hel.)
Fernandez, J. M. M., 692 (Lep.)
Fernández, L. J. & Fernández, R. F., 456 (Oph.)
Fernando, A. & De Ogampo, G., 51 (Y. & S.)
Fernando, C. F., 996 (Fev.)
Fernando, P. B., 828 (Hel.)
——, d'Silva, M., Stork, G. K. B. & Sinnatamby, G. R., 832 (Hel.)
Féron, J., 695 (Lep.)

Ferrabouc, L., Jaulmes, C. & Jude, A., 1032 (Leish.)
Ferrari, R. C., with Ivanissevich & Rivas, (717) (Hel.)
Ferreira, L. C., with Chagas, da Cunha, Deane, L., Deane, G., Guimarães, von Paumgartten & Sá, 446 (Leish.)
—— & Deane, L., 754 (Tryp.)
——, Mangabeira, O., Deane, L. & Chagas, A. W., 449 (Leish.)
Ferrer, I., 348 (Misc.)
Ferris, H. W. & Turner, T. B., 45, 47 (Y. & S.)
Fester, with Gavrilov & Dubois, 698 (Lep.)
Fialho, A., 507 (Misc.)
Ficker, M., 537, 538 (Lep.)
Field, J. W., 872 (B.R.)
——, Niven, J. C. & Guest, C., 498 (Mal.)
Figarella, J., with Rísquez & van Praag, 470 (Fev.)
Fimbel, M., with de Sèze & Laplane, 253 (Hel.)
Findlay, G. M., 350 (B.R.)
—— & MacCallum, F. O., 30, 635, 638 (Y.F.)
——, —— & Murgatroyd, F., 651 (Y.F.)
Finlayson, M. H., 867 (Vms.)
—— & Grobler, J. M., 861 (Vms.)
——, with Shapiro & Sapeika, 867 (Vms.)
Fisk, G. H., 591 (Hel.)
Fitzpatrick, F., with Zinsser & Wei, 1002 (Fev.)
Fitzpatrick, F. K., 998 (Fev.)
Flamini, S., 723 (Hel.)
Flarer, F., 454 (Leish.)
Flecker, H., 575 (Vms.)
Fletcher, A. E., with Jordan, 472, 990 (Fev.)
Flippin, H. F., 474 (Fev.)
Florand, J., with Huber, Lièvre & Néret, 123 (Bl.)
Foissin, J., with Loubeyre & Tillier, 296 (Am.)
da Fonseca, F., 636, 653 (Y.F.)
—— & Artigas, P., 652, 653 (Y.F.)
Fonseca, R. C., Kourí, P. & Basnuevo, J. G., 593 (Hel.)
Forkner, C. E., with Ch'u, 417 (Misc.)
Forro, M., with Dubois, 842 (Hel.)
Fort, P. C. L., with Laurens & Bernier, 1000 (Fev.)
Foster, A. O. & Johnson, C. M., 850 (Hel.)
Fourestier, M., with Sergent & Galliano, 716 (Hel.)
Fournials, 216, 667 (Tryp.)
Fournier, 988 (Fev.)
Fournier, J., 602 (Dys.), 895 (Chl.)
——, with Raynal, 983 *bis* (Fev.)
Foy, H., with Fairley, Bromfield & Kondi, 422 (Misc.)
—— & Kondi, A., 121, 122, 887 (Bl.)
Fraenkel-Conrat, H., with Slotta, 855 (Vms.)
Franca, M., with Shannon & Whitman, 27 (Y.F.)
Francis, E., 760 (R.F.)
Francke, M., with Ciuca, Balteanu, Alexa & Micu, 11 (Mal.)
Franks, A. G., with Astrachan, 670 (Tryp.)
Fraser, G., 422 (Misc.)
Fraser, P. K., 104 (Der.)
French, M. H., 215, 216, 747 (Tryp.)
Freund, L., 41 (Hel.)
Frickers, J., 156 (Hel.)
Friedrich, H., 252 (Hel.)
v. Friedrich, L., 603 (Dys.)
Fróes, H. P., (452) (Leish.)
Frye, W. W. & Meleney, H. E., 293 (Am.)
Fulton, J. D., with Christophers, 340 (Mal.)
Fulton, M. C., with Sydenstricker, Schmidt, New & Geeslin, 551 (Pel.)
Füsthy, O., 600 (Dys.)

G.

Gabaldón, A., 91 (B.R.), 821 (Mal.)
Gallais, with Jospin, 217 (Tryp.)
Gallais, P., (720) (Hel.)
Galliano, E. J., with Sergent & Fourestier, 716 (Hel.)
Galliard, H., 150, 328, (328), 837, 843 (Hel.)
Galli-Valerio, B., 603 (Dys.)
Galloway, I. A., 195 (Rab.)
Gallut, J., with Bernard & Guillerm, 373 *bis*, 374 (Chl.)
Gangolli, D. A., (718) (Hel.)
Ganguly, R., with Ray, 907 *bis* (Diet.)
Gaona, R. J., with Gatti, 696 (Lep.)
Garcia, E. Y., 138 (Mal.)
——, with de Jesus & Jao, 8 (Mal.)
Gardner, W. A. & Dexter, L., 783 (Mal.)
Garin, C., & Roman, 833 (Hel.)
Garnham, P. C. C., 492 (Mal.)
Garrett, E. B., 560 (Bb.)
Garzon, R., with Castellano, (558) (Pel.)
Gasic, G., 229 (Tryp.), 497, (680) (Mal.)
——, with Faiguenbaum, 497 (Mal.)
Gatti, C., Boggino, J. & Prieto, C., 1032 (Leish.)
—— & Gaona, R. J., 696 (Lep.)
Gaube, R., with Hillemand, 304 (Am.)
Gaud, 1001 (Fev.)
Gauducheau, A., 292 (Am.), (905) (Chl.)
Gaugeat, M., with Rousseau, 539, 1021 (Lep.)
Gaviña Alvarado, E. R., Negri, T. & Mosto, 106 (Der.)
Gavrilov, W., Bobkoff, G. & Laurencin, S., 929 (Mal.)
——, Dubois, A. & Fester, 698 (Lep.)
—— & Laurencin, S., 448 (Leish.)
Gear, J., 157 (Misc.), 997 (Fev.)
Gear, J. H. S., 996 (Fev.)
—— & de Meillon, B., 659 (Tryp.)
Geeslin, L. E., with Sydenstricker, Schmidt, Fulton & New, 551 (Pel.)
Geiman, Q. M., with Tyzzer, 598 (Dys.)
Gelperin, J., with Spies, Aring & Bean, 553 (Pel.)
Genevray, J., 904 (Chl.)
—— & Bruneau, J., 378 (Chl.)
——, —— & Seyberlich, A., 904 (Chl.)
——, Toumanoff, C. & Hoang-Tich-Try, 131 (Mal.)
Gentzkow, C. J. & Callender, G. R., 257 (Mal.)
——, with ——, 679 (Mal.)
Gerlach, F., 193 (Rab.)
Germain, A. & Morvan, A., 496 (Mal.)
Germond, R. C., 234 *ter* (Lep.)
Geronnez, E., with Schwetz, 777, 778 (Mal.)
Ghalioungui, P., with Alport, (556) (Pel.)
——, with —— & Hanna, 555 (556) (Pel.)

Ghermani, A., with Claudian, 546 (Pel.)
Ghidini, G. M., 214 (Tryp.)
Ghosal, S. C., with Lal & Mukherji, 896 (Chl.)
Ghose, A. K., (724) (Hel.)
Ghosh, B. N., 179 (B.R.)
—— & De, S. S., 856 (Vms.)
——, —— & Bhattacharya, D. P., 855 (Vms.)
Ghosh, H., 379 (Chl.)
Gibbes, J. H., 576 (Vms.)
Gibbins, E. G., (843) (Hel.)
Gibbons, R. J., 474 (Fev.), 967 (Pl.)
Gifford, M. A., with Dickson, 507 (Misc.)
Giglioli, G., 809 (Mal.), 940 (Misc.)
Gillet, R., (680) (Mal.)
Gimlette, J. D., 432 (B.R.)
Gioseffi, M., 347 (Misc.)
Giovannola, A., (623) (Hel.), 919, 930 (Mal.)
Girard, G., 310 (Pl.)
—— & Girard, M., 971 (Pl.)
—— & Robic, J., 314 (Pl.)
Giraud, P. & Bergier, P., 1026 *bis* (Leish.)
——, Cabassu, H. & Bergier, P., 1026 (Leish.)
—— & Poinso, R., 450 (Leish.)
Giroud, P., 982, 983 (Fev.)
—— & Panthier, R., 984, 994 (Fev.)
Giulio, B., 35 (Hel.)
Giunta, B. & d'Ignazio, C., 989 (Fev.)
Glotzer, S., 115 (Lept.)
Glover, R. E., 725 (Rab.)
Glueck, H. I., with Rachmilewitz, 552 (Pel.)
Gminder, E. J., 52 (Y. & S.)
Goarnisson, J., with Lefrou, 738 (Tryp.)
Godal, J., 104 (Der.)
Gohar, M. A., 471 (Fev.)
Gohar, N., 103 (Der.)
Goinard, P., 952 (B.R.)
de Goldfiem, A. S. & de Goldfiem, J. S., 697 (Lep.)
Goldman, D., 398 (Mal.)
Goldstein, F., with Manwell, 1011 (Mal.)
Gomes, J. M., 99 (Der.), 241 (Lep.)
Gómez López, L. & Luna, G., 716 (Hel.)
Gomi, T., with Kubo & Baba, 504 (Misc.)
Goodman, M. H., 349 (Misc.)
Goodwin, M. H., Jr., with Hill, 261 (Mal.)
Gopsill, W. L., 38, 841 (Hel.)
Gorbitz, G., 257 (Mal.)
Gordon, L. S., with Wright & Bozicevich, 846 (Hel.)
Gordon, R. M., with Davey, 582 (Mal.)
Gore, R. N., 396 (Mal.)
Goret, P. & Mariette, C., 195 (Rab.)
Gouget, R., (680) (Mal.)
Gouriou A. F. X., (141) (Mal.)
Goyal, R. K., with Chopra, Pasricha, Lal & Sen, 909 (Diet.)
Goyle, A. N., 898 (Chl.)
Gradle, H. S., 457 (Oph.) ,
Grant, H. M., with Spies & Huff, 557 (Pel.)
Grant, J. M., with Spies, Stone & Mc Lester, 555 (Pel.)
Grasso, R., 303 (Am.)
Gratch, I., with Negri, (135) (Mal.)
Grattan, H. W., 365 (Chl.)
Graziani, with Huard & Long, 296 (Am.)
Grecou, A., Ionescou, N. G. & Constantinescou, P., 552 (Pel.)
Green, J. G., 392 (Mal.)
Green, R., 382 (Chl.)
Greval, S. D. S., Chandra, S. N. & Woodhead, L. S. F., 935 (Misc.)
——, Lowe, J. & Bose, R., 537 (Lep.)
——, Sen Gupta, P. C. & Napier, L. E., 1030 (Leish.)
Grobler, J. M., with Finlayson, 861 (Vms.)
Gross, M., 420 (Misc.)
Grüneis, P., 604 (Dys.)
Guenev, C., with Pavlov, 749 (Tryp.)
Guest, C., 397 (Mal.)
——, with Field & Niven, 498 (Mal.)
Guibert, J. & Boscq, H., 216 (Tryp.)
Guilhaumou, F., with Tisseuil, 694 (Lep.)
Guillaumou, F., 535 (Lep.)
Guillerm, J., with Bernard & Gallut, 373 *bis* 374 (Chl.)
Guimarães, F. N., with Chagas, da Cunha, Ferreira, Deane, L., Deane, G., von Paumgartten, & Sá, 446 (Leish.)
Guimarães, L. R., 312 (Pl.)
Guindy, 366 (Chl.), 982 (Fev.)
Guinto, R. S., with Rodriguez, 238 (Lep.)
Gunawardana, S. A., with Nicholls, 590 (Hel.)
Gunewardene, S. R., 832 (Hel.)
Gunther, C. E. M., 478 (Fev.), 889 (Bl.)
—— & Schroeder, A. G., 1000 (Fev.)
Gupta, S. K., with DeMonte, 379 (Chl.)

H.

de Haas, 51 (Y. & S.)
——, with Eskey, 968 (Pl.)
Hackett, L. W., 676 (Mal.)
——, Russell, P. F., Scharff, J. W. & White, R. S., 820 (Mal.)
Hakansson, E. G., 302 (Am.)
Halberstaedter, L., 213 (Tryp.)
Hall, M. C., 425 (Misc.), 846 (Hel.)
Hamburger, F., 846 (Hel.)
Hanan, E. B. & Zurett, S., 101 (Der.)
Hanasaki, T., 718 (Hel.)
Hanna, G., with Alport & Ghalioungui, 555, (556) (Pel.)
Hare, C. C., 252 (Hel.)
Hare, K. P., 508 (Misc.)
Hargett, M. V., 646 (Y.F.)
Haring, A. T., with Manwell, 588 (Mal.)
Harley-Mason, R. J., 841 (Hel.)
Harris, L. J., 556 (Pel.)
Harrison, G. F., (238) (Lep.)
Harwood, P. D., with Underwood, 840 (Hel.)
Hasell, P. G., with Weathersbee, 137 (Mal.)
Hassan, A., 619 (Hel.)
v. Hasselbach, H., with Bühler, 715 (Hel.)
Hasselmann, C. M., 1018 (Lep.)
Hasseltine, H. E., 692 (Lep.)
Hassett, C. J., 365 (Chl.)
Hauer, A., 923 (Mal.)
Hawaii, Territory of, 973 (Pl.)
Hawking, F., 220, 660, 671, 742 (Tryp.), 767 (R.F.)
Hay, G. G., 415 (Misc.)
Hayashi, F., 531 (Lep.)
Hazebroek, F. E. A., with Müller & Essed, F. E. A., 99 (Der.)
Heckenroth, M., 51 (Y. & S.)
Heggie, J. F., with Stevenson, 252 (Hel.)

Hegner, R., 495 (Mal.)
—— & Eskridge, L., 338, 585 (Mal.)
—— & Wolfson, F., 584, 929 (Mal.)
Heilmann, K., 604 (Dys.)
Heinemann, H., 479 (Fev.)
Helman, J., 158 (Misc.)
Hénaff, with Julliard, (996) (Fev.)
Hennessey, R. S. F., 949 (Misc.)
Henrard, C., with van Hoof & Peel, 211 (Tryp.)
Henry, A. F. X., 141, (688) (Mal.)
Henry, C., 929 (Mal.)
Henry Lester Institute of Medical Research, Shanghai, China, 704 (B.R.)
Hérivaux, A., Roncin, P. & Dao Van Thai, 918 (Mal.)
Herman, C. M., 337 (Mal.)
Herzig, A., 985 (Fev.)
Hewitt, R., 336, 585, 929, 1009, 1010 (Mal.)
Higoumenakis, G. K., 453 (Leish.)
Hilferty, M. M., with Shattuck, Bequaert, Sandground & Clark, 607 (B.R.)
Hill, R. A. & Goodwin, M. H., Jr., 261 (Mal.)
Hill, R. B., (336) (Mal.)
Hillemand, P. & Gaube, R., 304 (Am.)
Hilmy, F., with Wakil, 482 (Fev.)
Hingst, H. E., 138 (Mal.)
Hinman, E. H., 334 (Mal.)
Hitz, S., 466 (Fev.)
Hiyeda, K. & Terada, B., 717 (Hel.)
Ho, C., 426 (Misc.)
Ho, E. A., 1027 (Leish.)
Hoang-Tich-Try, with Genevray & Toumanoff 131 (Mal.)
Hoare, C. A., 205 (Tryp.)
—— & Broom, J. C., 661 (Tryp.)
Hocking, K. S., with Potter, 921 (Mal.)
Hodgkin, E. P., (8) (Mal.)
Hodgson, C. H., with Brown, 299 (Am.)
——, with Poynton, 149, 839 (Hel.)
Hoekstra, M., with Schüffner & Walch-Sorgdrager, 18, 25 (Y.F.)
Hoeppli, R., Feng, L. C. & Chu, H. J., 35 (Hel.)
——, with —— & Chung, 1027 (Leish.)
Hoffman, S. J., with Blatt & Schneider, 196 (Rab.)
Hoffman, W. A., 619 (Hel.)
——, with Rodríguez-Molina, 40 (Hel.)
Hoffmann, C. C., (681) (Mal.)
Hoffmann, C., with Tchitchibabine, 580 (Mal.)
Holden, H. F., with Feldberg & Kellaway, 565 (Vms.)
Hong Kong, 511 (Rep.)
Hood, M. & Olson, S. W., 847 (Hel.)
Hoodless, D. W., 943 *bis* (Misc.)
van Hoof, L., Henrard, C. & Peel, E., 211 (Tryp.)
——, with Rodhain & Muylle, 928 (Mal.)
Hopkins, G. H. E., 1038 (B.R.)
Hopman, B. C., 58 (Sp.)
Hopper, M. E., with Lewis & Montgomery, 103 (Der.)
Horack, H. M., 730 (Rab.)
Höring, F. O., 636 (Y.F.)
Horn, A. E., 428 (Misc.)
Hoyne, A. L. & Wolf, A. A., (146) (Hel.)
Hsiung, T., 600 (Dys.)
Hsü, H. F., (595) (Hel.)
—— & Chow, C. Y., 144 (Hel.)
—— & Khaw, O. K., 41 (Hel.)
Hu, S. M. K., 835 *bis*, 836 (Hel.)
Huard, P., 843 (Hel.)
——, Long, M. & Graziani, 296 (Am.)
Huber, J., Florand, J., Lièvre, J. A. & Néret, 123 (Bl.)
Huff, C. G., 8, 1010 (Mal.)
Huff, N. E., with Spies & Grant, 557 (Pel.)
Hulshoff, A. A., 503 (Misc.)
Hussein, H. A., with Kirk & McKelvie, 457 (Oph.)
Hutton, E. L., with Sinton & Shute, 401, 583, 925 (Mal.)
Hynes, K. E., (508) (Misc.)

I.

d'Ignazio, C., with Giunta, 989 (Fev.)
Ihlenfeldt, G., 50 (Y. & S.)
Impallomeni, R., 621 (Hel.)
Innes, J. R., 533, 1018 (Lep.)
Inouye, H., with Shimokawa, (720) (Hel.)
International Labour Office, 351 (B.R.)
Ionesco, N. G. & Constantinesco, P., 548 (Pel.)
——, with Grecou & Constantinescou, 552 (Pel.)
Ionescu, P., with Ciuca, Badenski & Teriteanu, 823 (Mal.)
——, with Tupa, Ciuca & Badenski, 822 (Mal.)
Ipsen, J., 861 (Vms.)
Ismail, A. A. & Soliman, M. A., 886 (Bl.)
Ivanissevich, O., Ferrari, R. C. & Rivas, C. I., (717) (Hel.)
——, Piñero, T. A., Risolía, A. A. & Rivas, (717) (Hel.)
Iwata, S., 995 (Fev.)
Iyengar, M. O. T., 148 (Hel.), 1005 (Mal.)
Iyengar, N. K., Sehra, K. B. & Mukerji, B., 855 (Vms.)

J.

Jack, R. W., 673 (Tryp.), 1040 (B.R.)
Jackson, R. B., 411 (Misc.)
Jacob, V. P. with Russell & Ramachandra Rao, 1005 (Mal.)
Jacotot, H., Colson, M. & Le Roux, G., 196 (Rab.)
Jadfard, H., with Burnet & Larabi, 105 (Der.)
Jadin, J. & Arnaldi, E., 642 (Y.F.)
Jahn, G., with Boecker, 729 (Rab.)
Jahnel, F., 213 (Tryp.)
James, E. R., 972 (Pl.)
James, S. P., 18, 631 (Y.F.)
—— & Tate, P., 339 (Mal.)
Jandolo, E., 821 (Mal.)
Jansen, G., with Deane, 754 (Tryp.)
Jao, S. G., with de Jesus & Garcia, 8 (Mal.)
Jaswant Singh, with Afridi & Abdul Majid, 9 (Mal.)
Jauffret, R., 749 (Tryp.)

Jaulmes, C., with Ferrabouc & Jude, 1032 (Leish.)
Jelinek, A., 496 (Mal.)
Jelke, H., 59 (Sp.)
Jellison, W. L., 968 (Pl.)
Jensen, V. & Roth, H., 147 (Hel.)
Jerace, F., 410 (Misc.), 661 (Tryp.), 1034 (Leish.)
Jeremiah, R., 540 (Lep.)
de Jesus, P. I., Jao, S. G. & Garcia, E. Y., 8 (Mal.)
Johnson, C. M., 563 (Vms.)
——, with Foster, 850 (Hel.)
Johnson, D. W., 116 (Lept.)
Jolly, A. & Danglemont, F., 782 (Mal.)
—— & di Ruggiero, 50 (Y. & S.)
Jonchère, H., with Peltier, Durieux & Arquié, 637 (Y.F.)
Jones, M., 328 (Hel.)
Jonnesco, D., 194, 726, 727 (Rab.)
—— & Zugravesco, I., 726 (Rab.)
Jordan, C. F., 475 (Fev.)
Jordan, F. L. J., 123 (Bl.)
Jordan, H., 1038 (B.R.)
Jordan, J. H. & Fletcher, A. E., 472, 990 (Fev.)
Jörg, M. E., with Mazza & Feijóo, 227 (Tryp.)
Jorge, R., 351 (B.R.)
Joseph, J. J., 532 (Lep.)
Jospin & Gallais, 217 (Tryp.)
Journal of the American Medical Association, 571 (Vms.)
Journal of the Association of Medical Women in India, 905 (Chl.)
Journal of the Public Health Association of Japan, 367 (Chl.)
Joyeux, C. & Sautet, J., 110 (R.F.)
——, —— & Artaud, P., 506 (Misc.)
Jude, A., with Ferrabouc & Jaulmes, 1032 (Leish.)
Julianelle, L. A., Sory, R., Smith, J. E. & Lange, A. C., 457 (Oph.)
Julliard, J. & Hénaff, (996) (Fev.)
Junior, P., (62) (Sp.)
Justin-Besançon, L., with Villaret, Klotz & Sikorav, 543 (Pel.)

K.

Kallinich, W., with Lodenkämper, 371 (Chl.)
Kalmus, H. & Kostić, D. Y., 263 (Mal.)
Kaplan, S. S., with Culbertson, 148 (Hel.)
Katz, G., with Sawitz & Tobie, 830 (Hel.)
Kau, L. S. & Wu, K., (37), (42) (Hel.)
Kaufmann, O., 115 (Lept.)
Kaufmann, W., 319 (Hel.)
Kawamura, R. & Yamamiya, C., 1000 (Fev.)
Kedah, 161 (Rep.)
Keil, E., 697 (Lep.)
Kekhcher, O. M., 259 (Mal.)
Kelantan, 169 (Rep.)
Kellaway, C. H., 568 (Vms.)
——, with Feldberg, 564 (Vms.)
——, with —— & Holden, 565 (Vms.)
Keller, A. E., with Leathers & McPhaul, 592, 828 (Hel.)
Kelly, R. J., 568 (Vms.)
Kemp, H. A., 991 (Fev.)
Kenawy, M. R., 620 (Hel.)
Kennedy, W. P., 417 (Misc.)
Kent, M. L. & Stahnke, H. L., 865 (Vms.)
Kentenich, A., with Sioli & Boldt, 494 (Mal.)
Kerr, K. B., with Otto, 833 (Hel.)
Kervingant, M. & Baré, J., 1016 (Lep.)
Kessel, J. F. & Sinitsin, D., 599 (Dys.)
Kessler, W. R., with Culbertson, 750 (Tryp.)
Kesteven, H. L., 104 (Der.)
Kettelkamp, G. D., Murphy, P. & Trumpe, D., 347 (Misc.)
Khalil, M., 326 *ter* (Hel.)
Khan, J. S., 696 (Lep.)
Khargpur, B.-N. Railway, 1008 (Mal.)
Khaw, O. K., with Hsü, 41 (Hel.)
Kielland, J., 544 (Pel.)
Kikuth, W., 816 (Mal.)
—— & Mudrow, L., 337 (Mal.)
Kimura, T., 560 (Bb.)
Kin, S., with Suwo, 1021 (Lep.)
King, C. & de Rozario, K. A., 416 (Misc.)
King, H., Lourie, E. M. & Yorke, W., 221 (Tryp.)
King, W. V., 403 (Mal.)
Kingsbury, A. N., 128 (Mal.) 197 (Rab.), 382 (Chl.), 463 (Fev.)
Kinugasa, M., 624, 627 (Hel.)
Kirby-Smith, J. L., 106 (Der.)
Kirk, R., 445 (Leish.), 731 (Rab.), 761, 762 (R.F.)
——, McKelvie, A. R. & Hussein, H. A., 457 (Oph.)
Kirschner, A., with Southwell, 155 (Hel.)
Kirwan, E. O'G., 957 (Oph.)
Kitchen, S. F., 138, 811 (Mal.)
——, with Boyd, 492, 495, 497, 682 *bis*, 811 (Mal.)
——, with —— & Matthews, 922 (Mal.)
Klenerman, P., 38 (Hel.)
Kligler, I. J. & Bernkopf, H., 193, 724, 728 (Rab.)
von Klobusitzky, D. & König, P., 566, (854) (Vms.)
Klotz, H. P., with Villaret, Justin-Besançon & Sikorav, 543 (Pel.)
Knabe, K., 863 (Vms.)
Knott, J., 152 (Hel.)
——, with O'Connor, 151 (Hel.)
Kō, T., 322 (Hel.)
Kobayashi, H. & Yumoto, Y., 624 (Hel.)
Koeslan, 236 (Lep.)
Koesoemadilaga, R. M. R., 898 (Chl.)
Koga, S., with Otomo & Tanaka, 312 (Pl.)
Kolmer, J. A. & Rule, A. M., 749 (Tryp.)
Kolodny, M. H., 755 *ter* (Tryp.)
——, with Culbertson, 231 (Tryp.)
Komp, W. H. W., with Clark, 819 (Mal.)
Kondi, A., with Fairley, Bromfield & Foy, 422 (Misc.)
——, with Foy, 121, 122, 887 (Bl.)
König, P., with von Klobusitzky, 566, (854) (Vms.)
Koo, C. K., with Yang, Landauer & Lin, 960 (Pl.)
Kopciowska, L. & Nicolau, S., 195 (Rab.)
——, with ——, Cruveilhier, Truche & Viala, 195 (Rab.)
Korobkova, E., Favorissova, B. & Krainova, A., 967 (Pl.)

Kostić, D. & Antić, D., 924 (Mal.)
Kostić, D. Y., with Kalmus, 263 (Mal.)
Kourí, P., Basnuevo, J. G., Sotolongo, F. & Anido, V., 599, 605 (Dys.), 626 (Hel.)
—— & Doval, J. M., 720 (Hel.)
——, with Fonseca & Basnuevo, 593 (Hel.)
Kouwenaar, W., with Boers & Wolff, 102 (Der.)
——, with Meyers, 838 (Hel.)
Krainova, A., with Korobkova & Favorissova, 967 (Pl.)
Krishnan, B. G., with Aykroyd, (1021) (Lep.)
Krishnan, K. V., 449 (Leish.), 889 (Bl.)
Kritchevski, I. L., 13 (Mal.)
Kubo, M., 155 (Hel.)
—— & Baba, H., 504 (Misc.)
——, —— & Gomi, T., 504 (Misc.)
Kuczynski-Godard, M. H., 484 (Fev.)
Kühnau, W. W., 556 (Pel.)
Kumm, H. W., with Coggeshall, 406 (Mal.)
—— & Novis, O., 28 (Y.F.)
Kundu, K. P. & Pa How, U., 366 (Chl.)
Kunert, H., 622 (Hel.)
Kupper, W. H., with Boyd & Matthews, 493 (Mal.)
Kusch, E., with Milam, 140 (Mal.)

L.

Lafferre, 889 (Bl.)
—— & Rouanet, (1018) (Lep.)
Lagodsky, H., 743 (Tryp.)
——, with Launoy, 212 (Tryp.)
Lahiri, B. N., with Smith, McGuire & Stephens, 729 (Rab.)
Lahiri, M. N. & Das, P. C., 377 (Chl.)
——, with Pasricha & Das, 367 (Chl.)
Laigret, J. & Auburtin, P., 370 (Chl.), 468 (Fev.)
—— & Durand, R., 993 (Fev.)
Lal, with Ahmad & Roy, 908 (Diet.)
Lal, with Sen, Chopra, Pasricha & Goyal, 909 (Diet.)
Lal, with Sankaran, Mukherji & Roy, 910 (Diet.)
Lal, R. B., 449 (Leish.), 889 (Bl.), 907 (Diet.)
——, Ahmad, B. & Roy, S. C., 908 (Diet.)
——, Ghosal, S. C. & Mukherji, B., 896 (Chl.)
——, Mukherji, S. P., Roy, S. C. & Sankaran, G., 910 (Diet.)
—— & Roy, S. C., 909 (Diet.)
Lal, S., with Chopra, Pasricha, Goyal & Sen, 909 (Diet.)
Lambrichts, G., (839) (Hel.)
Lancaster, R. L., with Schretzenmayr, 424 (Misc.)
Lancet, 31 (Y.F.), 555 (Pel.)
Landauer, E., (310) (Pl.)
——, with Yang, Koo & Lin, 960 (Pl.)
Lane, C., 721 (Hel.)
Lange, A. C., with Julianelle, Sory & Smith, 457 (Oph.)
de Langen, C. D., 944 (Misc.)
——, Boswijk, J. C. & van Nieuwenhuizen, C. L. C., 545 (Pel.)
Langeron, M., 136 (Mal.)
Langston, W. C., with Shukers & Day, 424 (Misc.)
Laplane, L., with de Sèze & Fimbel, 253 (Hel.)
Lara, C. B., 533 (Lep.)
Larabi, M., with Burnet & Jadfard, 105 (Der.)
Lassman, P., 503 (Misc.)
Lataste, C., with Farinaud, Baccialone & Canet, 816 (Mal.)
——, Farinaud, M. E. & Nguyen-Van-Lien, 815 (Mal.)
——, Nguyen-Van-Lien & Farinaud, M. E., 815 (Mal.)
Launoy, L., 212, 744 (Tryp.)
—— & Lagodsky, H., 212 (Tryp.)
Laurencin, S., with Gavrilov, 448 (Leish.)
——, with —— & Bobkoff, 929 (Mal.)
Laurens, J. R., Fort, P. C. L. & Bernier, G. L., 1000 (Fev.)
Laurent, D., 949 (Misc.)
Lavier, G., 674 (Tryp.)
——, with Breton, 718 (Hel.)
Lavrinenko, N., with Ciuca, Ballif & Chelaresco, 11, 390, 924 (Mal.)
Law, F. W., 455 (Oph.)
Lawkowicz, W., 485 (Fev.)
——, with Anigstein, 993 (Fev.)
Lazar, C., with Mezincesco, Cornelson & Buşila, 11 (Mal.)
League of Nations, 892 (Chl.), 973 (Pl.)
Leahy, H. F., 1031 (Leish.)
Leathers, W. S., Keller, A. E. & McPhaul, W. A., 592, 828, (Hel.)
Le Bourdellès, B., 922 (Mal.)
Leccisotti, G., 461 (Fev.)
Lecercle, 723 (Hel.)
Le Chuiton, F. & Pennanéac'h, J., 465 (Fev.)
Ledentu, G. & Peltier, M., 408 (Misc.)
Leeson, H. S., (810) (Mal.)
van Leeuwen, H. C., 56 (Sp.)
Lefebvre, 389 (Mal.)
Lefèvre, 30 (Y.F.)
Lefrou, G. & Goarnisson, J., 738 (Tryp.)
Legezynski, S., 725 (Rab.)
Lehucher, P., with Anderson, (155) (Hel.)
Leite, M. R., 603 (Dys.)
Leitner, A. J., 534 (Lep.)
Lenti, P., with Fattovich, 262 (Mal.)
Lentjes, L. J. M., 923 (Mal.)
León, L. A., 720 (Hel.)
de Leon, W., with Africa, 589 (Hel.)
Lépine, P., 995 (Fev.)
——, with Cruveilhier & Viala, 197 (Rab.)
—— & Sautter, V., 728 (Rab.)
Leprosy Review, 531 (Lep.)
Le Roux, G., with Jacotot & Colson, 196 (Rab.)
Leslie, C. J., 252 (Hel.)
Lester, H. M. O., 204, 672 (Tryp.)
Lestoquard, F., 984 (Fev.)
——, with Sergent, Parrot & Donatien, 1026 (Leish.)
Levit, V., with Slatineanu, Balteanu & Nitulescu, 545 (Pel.)
——, with ——, ——, Sibi & Nitzulesco, 545 (Pel.)
Lewillon, R., 729 (Rab.)
Lewis, D. R., 557 (Pel.)
Lewis, G. M., Montgomery, R. M. & Hopper, M. E., 103, (Der.)

Lewis's Medical and Scientific Library, 182, 953 (B.R.)
Lewthwaite, R., 197 (Rab.), 463 (Fev.), 941 (Misc.)
Lida, E., with Marenzi, 417 (Misc.)
di Liddo, F., 243 (Lep.)
Liddo, S., 721 (Hel.)
Lieou, Y. C., with Raynal & Feissolle, 896 (Chl.)
Lièvre, J. A., with Florand, Huber & Néret, 123 (Bl.)
Lin, P. C., with Yang, Landauer & Koo, 960 (Pl.)
Lindeboom, G. A. & Wientjes, J. E. B., 60 (Sp.)
Lins, S. A., 28 (Y.F.)
Linton, R. W., Mitra, B. N. & Seal, S. C., 371 (Chl.)
——, Shrivastava, D. L., Seal, S. C. & Mookerji, S. P., 370 (Chl.)
Linton, W. S., 1031 (Leish.)
Lippelt, H., 596 (Dys.)
—— & Mohr, W., (839) (Hel.)
Lister, S., 196 (Rab.)
Litarczek, S. & Chisar, (717) (Hel.)
Liu, P. Y., with Zia, 472 (Fev.)
——, with —— & Pang, 992 (Fev.)
——, —— & Wang, K. C., 468 (Fev.)
Livadas, G., Canellakis, A. P. & Valaoras, V. G., 397 (Mal.)
Livierato, S., (261) (Mal.)
Lloverol, H., 749 (Tryp.)
Lloyd, B. J., 31 (Y.F.)
Lobanov, V. & Fedorov, V., 966 (Pl.)
Lobo, M. M., 228 (Tryp.)
Localio, S. A., with Dobriner & Strain, 547 (Pel.)
Lodenkämper, H. & Kallinich, W., 371 (Chl.)
Lodewyckx, A., 766 (R.F.)
Loe, F., 457 (Oph.)
Logan, C. E., with Traut, 157 (Misc.)
van Loghem, J. J., 375 (Chl.)
Lombard, R., with Dusan & Tivollier, (535) (Lep.)
——, with Roger & Boudouresques, (535) (Lep.)
Long, M., with Huard & Graziani, 296 (Am.)
Lorando, N., 992 (Fev.)
—— & Papanastassiou, E., 991 (Fev.)
Loubet, R., with Esnault, 453 (Leish.)
Loubeyre, J., Tillier, H. & Foissin, J., 296 (Am.)
Lourie, E. M., with King & Yorke, 221 (Tryp.)
—— & Yorke, W., 221 (Tryp.)
Loustau, H. L., with Talice & Sapriza, 227 (Tryp.)
Lowe, J., 235, 236, 1013, 1015 (Lep.)
—— & Chatterji, S. N., 534, 1015 (Lep.)
—— with Greval & Bose, 537 (Lep.)
Lucherini, T., 397 (Mal.)
Luna, G., with Gómez López, 716 (Hel.)
Lwoff, M., 447 (Leish.)
Lyon, M., 699 (Lep.)
Lyons, F. M., 455 (Oph.)

M.

Maassen, R., 546 (Pel.)
MacCallum, F. O., with Findlay, 30, 635, 638 (Y.F.)
MacCallum, F. O., with Findlay & Murgatroyd, 651 (Y.F.)
Macchiavello, A., 266 (B.R.)
—— & Contreras, M., (315) (Pl.)
Macdonald, G., 926 (Mal.), 939 (Misc.)
McGuire, J. P., with Smith, Stephens & Lahiri, 729 (Rab.)
McGusty, V. W. T., 943 (Misc.)
Macit Arif, 850 (Hel.)
Mackay, R., 387 (Mal.)
Mackehenie, D., 484 (Fev.)
McKelvie, A. R., with Kirk & Hussein, 457 (Oph.)
MacKenna, R. M. B., 704 (B.R.)
Mackenzie, D. L. & Bean, H., 599 (Dys.)
Mackie, D. B., with Stewart, 316 (Pl.)
Mackie, F. P. & Crabtree, H. S., 32 (Y.F.)
McKinley, E. B., 1020 (Lep.)
Mackinnon, J. E., 868 (Vms.)
McLean, J. A., 420 (Misc.)
McLester, J. B., with Spies, Grant & Stone, 555 (Pel.)
MacMahon, R., 259 (Mal.)
McNaught, J. B., 848 (Hel.)
MacNaughton, W. G., 532 (Lep.)
McPhaul, W. A., with Leathers & Keller, 592, 828 (Hel.)
McRobert, G. R., 422 (Misc.)
Macheboeuf, M. & Mandoul, R., 724 (Hel.)
Maeda, T., with Narihara, Yumoto & Osaka, 594 (Hel.)
——, with Shionuma & Nagai, 236 (Lep.)
Maegraith, B., 421 (Misc.)
de Magalhães, O., 478 *bis*, 998, 999 (Fev.), (575), 865, 866 (Vms.)
Magallon-Graineau, E., with Montestruc, de Balmas & Pignol, 770 (Lept.)
Magliano, G. & Azzi, E., 482 (Fev.)
Mahdi, M. A., with Shukry & El Gholmy, 912 (Diet.)
Mail, G. A., 577 (Vms.)
Mainzer, F., 544, (545) (Pel.), 39, 620 *bis* (Hel.)
Maitra, G. C., Sen Gupta, P. N. & Thant, U., 366 (Chl.)
Maitra, N. M., 900 (Chl.)
——, with Pandit, 376 (Chl.)
Majid, S. A., with Afridi, 678 (Mal.)
Majumdar, A. R., 267 (B.R.)
Majumdar, D. N., with Napier, 418 (Misc.)
Makar, N., 838 (Hel.)
Malaihollo, J. F., 694 (Lep.)
Malaya, 729 (Rab.)
Malik, K. S., with Chatterjee, 379 (Chl.)
Malycheva, A. N., 785 (Mal.)
Manalang, J., 539 *bis* (Lep.)
Manceaux, A., 812 (Mal.)
—— & Alcay, M., 626 (Hel.)
Mandoul, R., 724 (Hel.)
——, with Macheboeuf, 724 (Hel.)
Mandroux, J., with Denéchau, 771 (Lept.)
Mangabeira, O., with Ferreira, Deane, L. & Chagas, A. W., 449 (Leish.)
Mangalik, V., with Chaudhuri, 424 (Misc.)
Mannino, S., with l'Abbate, 111 (R.F.)
Mannozzi-Torini, M., 745 *bis* (Tryp.)
Manson, D., 1009 (Mal.)

Manson-Bahr, P., 157, 941 (Misc.), 295 (Am.), 521 (B.K.)
—— & Ransford, O. N., 550 (Pel.)
Manwell, R. D., 584 (Mal.)
—— & Goldstein, F., 1011 (Mal.)
—— & Haring, A. T., 588 (Mal.)
Maplestone, P. A. & Mukerji, A. K., 322 (Hel.)
Marangos, G., 251 (Hel.)
Marañon, G., 814 (Mal.)
Marchoux, E. & Chorine, V., 241, 540, 698 (Lep.)
—— & Prudhomme, R., 240 (Lep.)
Marenzi, A. & Lida, E., 417 (Misc.)
Mariani, G., 1001 (Fev.)
Marie-Suzanne, 695 (Lep.)
Mariette, C., with Goret, 195 (Rab.)
Marque, 457 (Oph.)
Marras, F. M., 373 (Chl.)
Marschall, F., 887 (Bl.)
Marseille, A., 145 (Hel.)
Marsh, F., 946 (Misc.)
Marshall, G. R. & Wood, Q. L., 329 (Hel.)
Marshall, J. F., 520 (B.R.)
Martial, J. E., 945 (Misc.)
Martin, D. S., 99 (Der.)
Martin, P., 604 (Dys.)
Martin del Campo, R., 853 (Vms.)
Martindale, 433 (B.R.)
Martins, A. V. & Dos Anjos, W. V., 618 (Hel.)
—— & Versiani, W., 39, 623 (Hel.)
Massani, R. M. M., 283 (Am.)
Masseguin, 220 (Tryp.)
Massias, C., 380 (Chl.), 506 (Misc.)
Mathew, M. I., 1005 (Mal.)
Mathew, R. Y., 469 (Fev.)
Mathieson, D. R. & Watson, B. A., 452 (Leish.)
Mathis, M., with Digonnet, 765 (R.F.)
——, with Roubaud & Colas-Belcour, 928 (Mal.)
Mattei, C., 301 (Am).
Matthews, C. B., with Boyd, 780, 809 (Mal.)
——, with —— & Kitchen, 922 (Mal.)
——, with —— & Kupper, 493 (Mal.)
Matthews, R. S., 551 (Pel.)
Mauzé, J., (834) (Hel.)
Mayfield, R. B., 311 (Pl.)
Maynard, N. H., 697 (Lep.)
Mayne, B. & Young, M. D., 139 (Mal.)
Mazza, S., 231 (Tryp.)
Mazza, S., *et al*, 752 (Tryp.)
——, Jörg, M. E. & Feijóo, E. J. C., 227 (Tryp.)
Measham, J. E., 393 (Mal.)
de Medina, F., with Talice & Rial, 225, 226 (Tryp).
Medulla, C., 573 (Vms.)
Megaw, J. W. D., with Rogers, 606 (B.R.)
Mehta, H., 247 (Lep.)
Meijer, F. H., 795 (B.R.)
Meijer, W. C. P., 717 (Hel.)
Meiller, E., with Romano & Rey, 605 (Dys.)
de Meillon, B., 135 (Mal.)
——, with Gear, 659 (Tryp.)
Meira, J. A., 53 (Sp.)
Melchior, E. & Özkan, A., 1007 (Mal.)
Meleney, H. E., with Frye, 293 (Am.)
Melik-Adamian, S. S., 262 (Mal.)
de Mello, I. F., 9, 396 (Mal.)
Mendes, A. B. C., 1028 (Leish.)
Mendheim, H., 588 (Hel.)
Menk, W. & Mohr, W., 786 (Mal.)
Menon, M. K., with Russell & Ramachandra Rao, 131 (Mal.)
Menon, T. B., 1029 (Leish.)
Mertens, W. K. & Mochtar, A., 371, 901 (Chl)
Messerlin, A., with Sicault, 395, 781 (Mal.)
Meunier, R., 132 (Mal.), 988 (Fev.)
Meyer, K. F., 315 *bis* (Pl.)
——, with Stewart, 101 (Der.)
Meyers, F. M., 558, 559 (Bb.)
—— & Kouwenaar, W., 838 (Hel.)
Mezincesco, D. & Cornelson, D. A., with Lazar, C. & Buşila, L., 11 (Mal.)
Mian, A. S., with Pasricha, de Monte & Chatterjee, 903 (Chl.)
Michelson, E. G., 403 (Mal.)
Micu, I., with Ciuca, Balteanu, Francke & Alexa, 11 (Mal.)
Milam, D. F. & Coggeshall, L. T., 583 (Mal.)
—— & Kusch, E., 140 (Mal.)
Millan, J. M. & Nespolo, J. F. V., 321 (Hel.)
Millen, R. M., with Thomas, 888 (Bl.)
Miller, M. J., with Faust, d'Antoni, Odom, Peres, Sawitz, Thomen, Tobie & Walker, 144 (Hel.)
Millischer, P., 200 (Rab.)
Minec, with Robic, 313 (Pl.)
Mingardon, G., with Olmer & Montagnier, 1030 (Leish.)
Mirra, G., 488 (Mal.)
Mirzojan, N. A., 1033 (Leish.)
Missiroli, A., 391, 919 (Mal.)
Mitchell, J. P., 1038 (B.R.)
Mitra, B. N., with Linton & Seal, 371 (Chl.)
——, with Seal, 897 (Chl.)
Mitra, K., 9 (Mal.)
Mitra, P. N., 834 (Hel.)
Miyajima, M., 623 (Hel.)
Mochtar, A. & Baars, J. K., 371 (Chl.)
—— & Esseveld, H., (770) (Lept.)
——, with ——, 115 (Lept.)
——, with Mertens, 371, 901 (Chl.)
——, with Sardjito & Tjitrohoepojo, 235 (Lep.)
Mody, S. H., 574 (Vms.)
Mohan, B. N., with Russell, 1004 (Mal.)
Mohr, W., with Lippelt, (839) (Hel.)
——, with Menk, 786 (Mal.)
Moise, R., 829 (Hel.)
Moise, R. M. R., 108 (R.F.)
Moisesco, T., Paidiu, V. & Dumitresco, D., 717 (Hel.)
Molina, A., 198 (Rab.)
Mollaret, with Breuil, 331 (Mal.)
Mollaret, P. & Bonnefoi, A., 774 (R.B.F.)
—— & Schneider, J., 817 (Mal.)
Mollari, M. & Anzulovic, J. V., 600 (Dys.)
Mollow, W., (717) (Hel.)
Molser, H., 841 (Hel.)
Montagnier, L., with Olmer & Mingardon, 1030 (Leish.)
de Monte, A. J. H., with Pasricha, Chatterjee & Mian, A. S., 903 (Chl.)
Montel, L. R., 349 (Misc.)
Montenegro, J., 641 (Y.F.)

Montestruc, E., 948 (Misc.)
——, de Palmas, M., Pignol, A. & Magallon-Graineau, E., 770 (Lept.)
Montgomery, R. M., with Lewis & Hopper, 103 (Der.)
Monti, G., with Poggi, 1033 (Leish.)
Mookerji, S. P., with Linton, Shrivastava & Seal, 370 (Chl.)
de Moor, C. E., 374 (Chl.)
Moore, M., 101 (Der.)
Morales Muñoz, T., Bernal Londoño, M. & de Souza Araújo, H. C., 690 (Lep.)
Moreau, P., 332 (Mal.)
Moreau, R. E., 664 (Tryp.)
Morénas, L., 432 (B.R.)
Morgan, E. L., with Bearup, 721 (Hel.)
Morgan, M. T., 313 (Pl.)
Morioka, K., 427 (Misc.)
Morisita, T., 763 (R.F.)
Morrow, G., Syverton, J. T., Stiles, W. W. & Berry, G. P., 772 (Lept.)
Morvan, A., with Germain, 496 (Mal.)
Möschl, H., 938 (Misc.)
Moshkovski, S. D. & Syrkine, S. A., 932 (Mal.)
Mosna, E., 927 (Mal.)
Moss, E. S., with Schenken, 845 (Hel.)
Mosto, D., with Gaviña Alvarado & Negri, 106 (Der.)
Motais, F., 956 (Oph.)
Motta, J., 534 (Lep.)
de Moura, A. M., 237 (Lep.)
Mudaliar, A. L., 180 (B.R.)
Mudrow, L., with Kikuth, 337 (Mal.)
Mueller, J. F., 250 (Hel.)
Muench, H., with Saunders, 44 (Y. & S.)
Muggleton, W. J., 521 (B.R.)
Muir, E., 1018 (Lep.)
Mukerji, A. K., with Maplestone, 322 (Hel.)
Mukerji, B., with Iyengar & Sehra, 855 (Vms.)
—— with Lal & Ghosal, 896 (Chl.)
Mukherji, S. P., with Lal, Roy & Sankaran, 910 (Diet.)
Müller, H., Essed, W. F. R. & Hazebroek, F. E. A., 99 (Der.)
Mulligan, H. W. & Afridi, M. K., 821 (Mal.)
Murano, G. & Vecchio, F., 1029 (Leish.)
Murdock, J. R., 969 (Pl.)
Murgatroyd, F., 771 (Lept.)
——, with Findlay & MacCallum, 651 (Y.F.)
Murphy, P., with Kettelkamp & Trumpe, 347 (Misc.)
Muto, S., 624 (Hel.)
Muylle, G., with Rodhain & van Hoof, 928 (Mal.)
Myers, V. C. & Eddy, H. M., 933 (Misc.)

N.

Nagai, K., with Shionuma & Maeda, 236 (Lep.)
Nair, V. G. & Pandalai, N. G., 536 (Lep.)
Nandi & Dikshit, 1008 (Mal.)
Napier, L. E., 422, 935 (Misc.), 542 (Pel.), 1030 (Leish.)
——, with Greval & Sen Gupta, 1030 (Leish.)
—— & Majumdar, D. N., 418 (Misc.)
——, with Sen Gupta, Rai Chaudhuri & Chaudhuri, 543 (Pel).
Narihara, N., 601 (Dys.)
——, Yumoto, Y., Osaka, K. & Maeda, T., 594 (Hel.)
Nash, T. A. M., 224 (Tryp.)
Nastasi, A., 618 (Hel.)
Nataf, R., with Burnet & Cuénod, 955 (Oph.)
Naudi, J., (469) (Fev.)
Negri, T., with Gaviña Alvarado & Mosto, 106 (Der.)
Néret, with Huber, Florand & Lièvre, 123 (Bl.)
Neri, F. & Gratch, I., (135) (Mal.)
Nespolo, J. F. V., with Millan, 321 (Hel.)
Neuhaus, W., (628) (Hel.)
New, J. S., with Sydenstricker, Schmidt, Fulton & Geeslin, 551 (Pel.)
Nguyen-Dinh-Hao, 132 (Mal.)
Nguyen-van-Huong, with Souchard, 102 (Der.)
Nguyen-van-Lien, with Farinaud & Lataste, 815 (Mal.)
——, with Lataste & Farinaud, 815 (Mal.)
Nguyen-Viem-Hai, 366 (Chl.)
Nicholls, L. & Gunawardana, S. A., 590 (Hel.)
Nicolau, S., 635 (Y.F.), 725 (Rab.)
——, Cruveilhier, L., Truche, C., Kopciowska, L. & Viala, C., 195 (Rab.)
——, with Kopciowska, 195 (Rab.)
Nicolle, P. & Simons, H., 746 (Tryp.), 933 (Misc.), 984 (Fev.)
Nietro Roaro, D. & Caballero, E., 624 (Hel.)
van Nieuwenhuizen, C. L. C., with de Langen & Boswijk, 545 (Pel.)
Nigeria, 736 (Tryp.)
Nishimura, H., 367 (Chl.)
Nitzulesco, I., with Slatineanu, Balteanu, Sibi & Levit, 545 (Pel.)
Nitulescu, J., with ——, —— & Levit, 545 (Pel.)
Niven, J. C., 399 (Mal.)
——, with Field & Guest, 498 (Mal.)
Nolan, M. O. & Reardon, L., 845 (Hel.)
Noury, M., with Blanc, 982 (Fev.)
Novis, O., with Kumm, 28 (Y.F.)
de Nunno, R., 500 (Mal.)
Nyasaland Protectorate, 71 (Rep.), 407 (Misc.)

O.

Oberdörffer, M., 532, 1021 (Lep.)
O'Brien, A. J. R., 18, 28 (Y.F.)
Ocampo, E., 228 (Tryp.)
O'Connor, F. W. & Knott, J., 151 (Hel.)
Odom, V., with d'Antoni, 829 (Hel.)
——, with Faust, d'Antoni, Miller, Peres, Sawitz, Thomen, Tobie & Walker, 144 (Hel.)
d'Oelsnitz, 451 (Leish.)
——, Saurin, Raibaudi & Daniel, 451 (Leish.)
Oesterlin, M., 181 (B.R.)
de Ogampo, G., with Fernando, 51 (Y. & S.)
Okabe, K., 40 (Hel.)
Oliver, A. G. & Oliver, J., 835 (Hel.)
Olmer, D. & Olmer, J., 473 (Fev.)
Olmer, J., Montagnier, L. & Mingardon, G., 1030 (Leish.)
Olson, S. W., with Hood, 847 (Hel.)

Olzscha, R., 364 (Chl.)
Omar, W., 375, (381) (Chl.)
Orenstein, A. J., (941) (Misc.)
Osaka, K., (623) (Hel.)
——, with Narihara, Yumoto & Maeda, 594 (Hel.)
Otomo, T., Koga, S. & Tanaka, I., 312 (Pl.)
Otsubo, G., (379) (Chl.)
Otten, L., 899 *bis* (Chl.)
Otto, G. F., with v. Brand, 325 (Hel.)
—— & Kerr, K. B., 833 (Hel.)
Otto, J. H. F., Tschan, T. J. & Au, L., 41 (Hel.)
Outes, J. D., 228 (Tryp.)
Overbeek, J. G., with Siegenbeek van Heukelom, 258 (Mal.)
Özkan, A., with Melchior, 1007 (Mal.)

P.

Pa How, U., with Kundu, 366 (Chl.)
Paidiu, V., with Moisesco & Dumitresco, 717 (Hel.)
Pakenham-Walsh, R. & Rennie, A. T., 12 (Mal.)
Palestine, 729 (Rab.)
Palinacci, A., with Saleun & Ceccaldi, 32 (Y.F.)
Pallary, P., (865) (Vms.)
Pallister, R. A., (561) (Bb.), 1030 (Leish.)
de Palmas, M., with Montestruc, Pignol & Magallon-Graineau, 770 (Lept.)
Palmer, L., 109 (R.F.)
Palmer, M. R., with Faget & Sherwood, 260 (Mal.)
Pampana, E. J., 410 (Misc.)
Pandalai, N. G., with Nair, 536 (Lep.)
Pandit, C. G., with Shortt & Rao, 483 (Fev.)
Pandit, S. R. & Maitra, N. M., 376 (Chl.)
Pang, K. H., with Zia & Liu, 992 (Fev.)
Panthier, R., with Giroud, 984, 994 (Fev.)
Paoliello, A., with Smith & Penna, 648 (Y.F.)
Papageorgiu, S., 773 (Lept.)
Papanastassiou, E., with Lorando, 991 (Fev.)
Papantonakis, E., 1028 (Leish.)
Paras, E. M., 245 (Lep.)
Pardo-Castelló, V., 45 (Y. & S.)
Parisi, E., 124 (Bl.)
Parker, R. R. & Davis, G. E., 479 (Fev.)
——, with Davis, 477 (Fev.)
——, with Philip, 466 (Fev.)
Parkin, B. S., 219 (Tryp.)
Parmakson, P., 239 (Lep.)
Parrot, L. & Catanei, A., 388 (Mal.)
——, ——, Ambialet, R. & Clastrier, J., 812 (Mal.)
——, —— & ——, with ——, (688) (Mal.)
——, with Sergent, E., Donatien & Lestoquard, 1026 (Leish.)
Pascale, H., with Pessôa, 323 *ter* (Hel.)
Pasqual, J. R. H., (563) (Vms.)
Pasricha, C. L., Chatterjee, D. N. & Das, P. C., 377 (Chl.)
——, —— & Paul, B. M., 383, 902 (Chl.)
——, with Chopra, Goyal, Lal & Sen, 909 (Diet.)
——, with Goyal, Chopra, Lal & Sen, 909 (Diet.)
Pasricha, C. L., Lahiri, M. N. & Das, P. C., 367 (Chl.)
——, with Lal, Chopra, Goyal & Sen, 909 (Diet.)
——, DeMonte, A. J. H. & Chatterjee, B. C., 379 (Chl.)
——, ——, —— & Mian, A. S., 903 (Chl.)
——, with Sen, Chopra, Goyal & Lal, 909 (Diet.)
Passa, 314 (Pl.)
Patiño Camargo, L., 25 (Y.F.)
Paul, B. M., with Pasricha & Chatterjee, 383, 902 (Chl.)
de Paula e Silva, G. S., 599, 603 (Dys.), 843 (Hel.)
Pauli-Magnus, 853 (Vms.)
Paulson, M. & Andrews, J., 294 (Am.)
Pauly, R. & Deprecq, R., 546 (Pel.)
von Paumgartten, M. J., with Chagas, da Cunha, Ferreira, Deane, L., Deane, G., Guimaraes, & Sá, 446 (Leish.)
Pavlov, P. & Guenev, C., 749 (Tryp.)
Pavlova, E. A., 284, 286, 287 (Am.)
Pawan, J. L., 726 (Rab.)
Pearson, G. H. & Yang, C. T., 829 (Hel.)
Peaston, H. & Renner, E. A., 926 (Mal.)
Peel, E., with van Hoof & Henrard, 211 (Tryp.)
Peery, T. M., 831 (Hel.)
Peiris, M. V. P., 298 (Am.)
Pellettieri, L., 1035 (Leish.)
Pellicciotta, R., 421 (Misc.)
Peltier, 650 (Y.F.)
——, Durieux, C., Jonchère, H. & Arquié, E., 637 (Y.F.)
——, with Ledentu, 408 (Misc.)
Penna, H. A., with Smith & Paoliello, 648 (Y.F.)
Penna, O., 199 (Rab.)
Pennanéac'h, J., with Le Chuiton, 465 (Fev.)
Penso, G., (666) (Tryp.)
Pepeu, F., 854 (Vms.)
Peragallo, I., 16 (Mal.)
Perdomo Hurtado, B., Ruiz Rodríguez, J. M. & Aníbal Osuna, 37 (Hel.)
Pereira, P. C. R., 242 (Lep.)
Peres, C., with Faust, d'Antoni, Odom, Miller, Sawitz, Thomen, Tobie & Walker, 144 (Hel.)
Perlis, 166 (Rep.)
Pesce, H., 59 (Sp.)
Pessôa, S. B. & Pascale, H., 323 *ter* (Hel.)
Petrov, M. I., 721 (Hel.)
Petrov, V. P., 794 (B.R.)
Petzetakis, M., 470 (Fev.)
Philip, C. B. & Dias, E., 998 (Fev.)
—— & Parker, R. R., 466 (Fev.)
Philipsz, G. L. C., with Veeraraghavan, 193 (Rab.)
Phisalix, (570) (Vms.)
Phisalix, M., 854 (Vms.)
Pieri, Sardou & Boudouresque, 295 (Am.)
Piéri, J., 472 (Fev.)
Pifano, F., 753 (Tryp.), 1035 (Leish.)
Pignol, A., with Montestruc, de Palmas & Magallon-Graineau, 770 (Lept.)
Pigoury, L. & Bernard, M., 985 (Fev.)
Pijper, A., with Broderick, 659 (Tryp.)
—— & Crocker, C. G., 468 (Fev.)

Pilcher, L. S., 474 (Fev.)
Pimentel-Imbert, M. F., with Carrión, 99 (Der.)
Pinard, M., Brumpt, L. & Raymondeau, M., 667 (Tryp.)
Piñero Garcia, P. P., 532 (Lep.)
Piñero, T. A., with Ivanissevich, Risolía & Rivas, (717) (Hel.)
Pinkerton, F. J., 237 (Lep.)
Pinkerton, H. & Sellards, A. W., 242 (Lep.)
——, with ——, 242 (Lep.)
Pinto, C., 1006 (Mal.)
Pinto, G. de S., 680 (Mal.)
Pires, J. R., 722 (Hel.)
Pistoni, F., 311 (Pl.)
Pittaluga, G., 484 (Fev.)
Pizzillo, G., 12, 400 *bis*, 685, 813 (Mal.)
Plaut, A. B. J., 938 (Misc.)
Poggi, I. & Monti, G., 1033 (Leish.)
Poindexter, H. A., 749 (Tryp.)
Poinso, R., with Giraud, 450 (Leish.)
Pokrovsky, S. N. & Zima, G. G., 589 (Hel.)
Poleff, L., 903 (Chl.), 956 (Oph.)
Pollitzer, R., with Robertson, 893 (Chl.)
Pontoni, L., 601 (Dys.)
Pool, R. M., with Walsh, 428 (Misc.)
Pooth, P., 1014 (Lep.)
Pop, A. & Budac, O., 743 (Tryp.)
Porges, O., 604 (Dys.)
Porte, 454 (Leish.)
Porter, A., 951 (B.R.)
Portier, with Sarrouy, 1007 (Mal.)
Portugal, H., 534 (Lep.)
Postic, S., 956 (Oph.)
Potapov, V. D., with Tiflov, 973 (Pl.)
Potop, I., with Slatineanu, (469) (Fev.), 549, 550 (Pel.)
Potter, C. & Hocking, K. S., 921 (Mal.)
Pottier, R., with Van den Branden, 671 (Tryp.)
Pouhin, E., 913 (Diet.)
Power, D'A., 432 (B.R.)
Poynton, J. O. & Hodgkin, E. P., 149, 839 (Hel.)
van Praag, A., with Risquez & Figarella, 470 (Fev.)
Prakken, J. R., with Carol, (51) (Y. & S.)
Pridie, E. D., 28 (Y.F.)
Prieto, C., with Gatti & Boggino, 1032 (Leish.)
Proske, H. O. & Watson, R. B., 813 (Mal.)
Provost, A., with Deschiens, 305 (Am.)
——, with Roubaud, 746 (Tryp.)
Prudhomme, R. O., 240, 698, (699 *bis*) (Lep.), 784 (Mal.)
——, with Marchoux, 240 (Lep.)
Public Health Reports, 646 (Y.F.)
Purcell, F. M., 701 (B.R.)
Pyman, F. L., 303 (Am.)

Q.

Quenardel, 384 (Chl.)

R.

Rabello, Jr., 239 (Lep.)
Rabello, E. & Rabello, Jr., 239 (Lep.)
Rabie, D., with Trabaud, 300 (Am.)
Rachmilewitz, M. & Glueck, H. I., 552 (Pel.)
Radianov, A., with Ciuca, Balteanu, J., Alexa, Balteanu, R. & Boeriu, 11 (Mal.)
——, with ——, ——, ——, Boeriu, Rugina & Stretcu, 11 (Mal.)
Radna, R., 536 *bis*, 694, 1021, 1022 *bis*, (Lep.), 666 (Tryp.)
Raffaele, G., 931 (Mal.)
Rafyi, A., with Delpy, 757 (R.F.)
Ragazzi, G., 505 (Misc.)
Raghavender Rao, S., 307 (Pl.)
Ragiot, C., with Farinaud, 786 (Mal.)
Raibaudi, with d'Oelsnitz, Saurin & Daniel, 451 (Leish.)
Rai Chaudhuri, M. N., with Sen Gupta, Chaudhuri & Napier, 543 (Pel.)
Raja, K. C. K. E., 365 (Chl.)
Rajagopalan, G., with Cochrane, 238 (Lep.)
Rajahram, S. G., 321 (Hel.)
Ramachandra Rao, with Russell & Menon, 131 (Mal.)
——, with —— & Jacob, 1005 (Mal.)
Ramakrishna, V., with Rao, 1039 (B.R.)
Ramijean, R., with Farinaud & Favot, 679 (Mal.)
Ramos Díaz, A., 310 (Pl.)
Ramos e Silva, J., 328 (Hel.)
Randall, R., with Simmons, Callender, Curry & Schwartz, 874 (B.R.)
Ransford, O. N., with Manson-Bahr, 550 (Pel.)
Rao, M. S., 963 (Pl.)
Rao, R. S., with Shortt & Pandit, 483 (Fev.)
Rao, S. R., 962 (Pl.)
Rao, V. V., & Ramakrishna, V., 1039 (B.R.)
Ravara, A., (261) (Mal.)
Ravn, J., 552 (Pel.)
Ravold, A., with Cowdry, 241 (Lep.)
Ray, with Ganguly, 907 *bis* (Diet.)
Ray, S. N. & Ganguly, R., 907 *bis*.
Raymondeau, M., with Pinard & Brumpt, 667 (Tryp.)
Raynal, J., 464 (Fev.)
—— & Fournier, J., 983 *bis* (Fev.)
——, Lieou, Y. C. & Feissolle, L., 896 (Chl.)
Read, W. D. B., 894 (Chl.)
——, Singh, G., Seal, S. C. & Bose, S., 895 (Chl.)
Reardon, L., 328, 329 (Hel.)
——, with Cram, 845 (Hel.)
——, with Nolan, 845 (Hel.)
Rebelo, A., with Soeiro, 488 (Mal.)
Redaelli, P., with Ciferri, 601 (Dys.)
Regules, U., with Talice & Alambarri, 226, 227 (Tryp.)
Remlinger, P., 727 (Rab.)
—— & Bailly, J., 194, 195, 196, 200 *bis*, 726 *bis*, 727, 731, 732 (Rab.)
Renner, E. A., with Peaston, 926 (Mal.)
Rennie, A. T., with Pakenham-Walsh, 12 (Mal.)
van Rensburg, S. W. J., 222 (Tryp.)
Reuter, J., with van Thiel, Sautet & Bevere, 1004 (Mal.)
Rey, S., with Romano & Meiller, 605 (Dys.)
Reyes Oribe, H., 455 (Leish.)
Reynes, V. & Trinquier, E., 667 (Tryp.)
Rhoads, C. P., with Barker, 57 (Sp.)
Rial, B., with Talice, 754 (Tryp.)
——, with —— & de Medina, 225, 226 (Tryp.)

Richard, A., 257 (Mal.)
Richards, A. I., 1037 (B.R.)
van Riel, J., 787 (Mal.)
Rietschel, 61 (Sp.)
Riou, M., with Vogel, 870 (Misc.), 905 (Chl.), 975 (Pl.), 1017 (Lep.)
van Riper, W., with d'Amour & Becker, 571 (Vms.)
Risolía, A. A., with Ivanissevich, Piñero & Rivas, (717) (Hel.)
Rísquez, J. R., Figarella, J. & van Praag, A., 470 (Fev.)
Rísquez, R., 716 (Hel.)
Ristorcelli, A., 312 (Pl.)
——, with Callot, 136 (Mal.)
Rivas, C. I., with Ivanissevich & Ferrari, (717) (Hel.)
——, with ——, Pinero & Risolia, (717) (Hel.)
Rivoalen, A., (425) (Misc.)
Rivoalen, P., with Tisseuil, 693 (Lep.)
Roberts, J. I., 965 *bis* (Pl.)
Robertson, R. C. & Pollitzer, R., 893 (Chl.)
Robic, J., 314 (Pl.)
——, with Girard, 314 (Pl.)
—— & Minec, 313 (Pl.)
Robineau, 648 (Y.F.)
Robinow, M. & Carroll, T. B., 577 (Vms.)
Robinson, G. G. (921) (Mal.)
Robinson, J. T., 445 (Leish.)
da Rocha Lima, H., 29 (Y.F.)
Rockefeller Foundation, 86 (B.R.)
Rodenwaldt, E., 178 (B.R.)
Rodhain, J., 931, 1011 (Mal.)
——, with van Hoof, L. & Muylle, G., 928 (Mal.)
Rodriguez, J. N. & Guinto, R. S., 238 (Lep.)
Rodriguez-Molina, R. & Hoffman, W. A., 40 (Hel.)
Rodriguez Olleros, A., 61 (Sp.)
Roger, H. & Boudouresques, J., 784 (Mal.)
——, —— & Lombard, R., (535) (Lep.)
Rogers, J. S. Y. & Tudhope, G. R., 253 (Hel.)
Rogers, L. & Megaw, J. W. D., 606 (B.R.)
Rohr, K., 55 (Sp.)
Rollé, R., with Valette, 304 (Am.)
Roman, with Garin, 833 (Hel.)
Roman, E., 720 (Hel.)
Romaña, C., 731 (Rab.), 756 (Tryp.)
Romano, N., Rey, S. & Meiller, E., 605 (Dys.)
Rome : Comitato Romano per l'Assistenza Antimalarica, 431 (B.R.)
Romeo, V., 261 (Mal.)
Roncin, P., with Hérivaux & Dao Van Thai, 918 (Mal.)
Rosenfeld, G., (598) (Dys.)
Roseveare, G. M., 1008 (Mal.)
Rosier, H. J., (315), 974 (Pl.)
Ross, G. A. P., 643 (Y.F.)
Ross, H., 246 (Lep.)
Rotberg, A., 242 (Lep.)
Roth, H., 146, 147 (Hel.)
——, with Jensen, 147 (Hel.)
Rouanet, with Lafferre, (1018) (Lep.)
Roubaud, E., 919 (Mal.)
——, Colas-Belcour, J. & Mathis, M., 928 (Mal.)
——, —— & Stefanopoulo, G. J., 638 (Y.F.)
—— & Deschiens, R., 833, (833) (Hel.)
—— & Provost, A., 746 (Tryp.)
Roudabush, R. L., (967) (Pl.)
Rousseau, G., 670 (Tryp.)
Rousseau, P. & Gaugeat, M., 539, 1021 (Lep.)
Row, R., 537 (Lep.)
Roy, with Ahmad & Lal, 908 (Diet.)
Roy, A. C., 566 (Vms.)
—— & Chopra, R. N., 566 (Vms.)
Roy, B. M., 503 (Misc.)
Roy, D. N., 336, 789 (Mal.)
——, Chandra, S. N. & Siddons, L. B., 681 (Mal.)
Roy, S. C., 255 (Hel.)
——, with Lal, 909 (Diet.)
——, with —— & Ahmad, 908 (Diet.)
——, with ——, Mukherji & Sankaran, 910 (Diet.)
——, with Sankaran, Lal & Mukherji, 910 (Diet.)
Rozario, K. A., with King, 416 (Misc.)
Ruffin, J. M. & Smith, D. T., 551 (Pel.)
Ruge, H., 1003 (Mal.)
di Ruggiero, with Jolly, 50 (Y. & S.)
Rugina, I., with Ciuca, Balteanu, Alexa, Boeriu, Stretcu & Radianov, 11 (Mal.)
Ruiz Castañeda, M., 994 (Fev.)
Ruiz Rodríguez, J. M., with Perdomo Hurtado & Aníbal Osuna, 37 (Hel.)
Rule, A. M., with Kolmer, 749 (Tryp.)
Russell, A. J., H., 256 (Mal.)
Russell, B. A. S., 130 (Mal.)
Russell, P. F., 787 (Mal.)
——, with Hackett, Scharff & White, 820 (Mal.)
——, Menon, M. K. & Ramachandra Rao, T., 131 (Mal.)
—— & Mohan, B. N., 1004 (Mal.)
——, Ramachandra Rao, T. & Jacob, V. P., 1005 (Mal.)
Russo, C., 368, 369 *bis* (Chl.), 964, 965 (Pl.)
Ryrie, G. A., (238), 247, 1018 (Lep.)

S.

Sá, B., with Chagas, da Cunha, Ferreira, Deane, L., Deane, G., Guimarães & von Paumgartten, 446 (Leish.)
Sabino, S. C., 563 (Vms.)
Sacchi, P., 866 (Vms.)
van Saceghem, R., 748 (Tryp.)
Sack, G., 139 (Mal.)
Saint Etienne, J., 421 (Misc.)
Salah, M., 51 (Y. & S.), 54 (Sp.)
Saleun, G., 632 (Y.F.), 658 (Tryp.)
——, Ceccaldi, J. & Palinacci, A., 32 (Y.F.)
Sambuc, E., (535) (Lep.)
Sánchez Posada, E., with Varela, 866 (Vms.)
Sandground, J. H., with Shattuck, Bequaert, Hilferty & Clark, 607 (B.R.)
Sang, J. H., 321 (Hel.)
Sangiorgi, G., 598 (Dys.)
Sankaran, G., with Lal, Mukherji & Roy, 910 (Diet.)
Sanner & Destribats, 483 (Fev.)
Sapeika, N., with Shapiro & Finlayson, 867 (Vms.)
Sapriza, J. P., with Talice & Loustau, (227) (Tryp.)

Sarcinella, F., 888 (Bl.)
Sardjito, M., Mochtar, A. & Tjitrohoepojo, M. S. H., 235 (Lep.)
Sardou, with Pieri & Boudouresque, 295 (Am.)
Sarikjan, S. J., 677 (Mal.)
Sarkar, S. K., 981 (Fev.)
Sarkissjan, M. A., 292 (Am.)
Sarles, M. P., (156) (Hel.)
Sarrouy & Portier, 1007 (Mal.)
Saunders, G. M. & Muench, H., 44 (Y. & S.)
Saurin, with d'Oelsnitz, Raibaudi & Daniel, 451 (Leish.)
Sautet, J., 296 (Am.)
——, with Joyeux, 110 (R.F.)
——, with —— & Artaud, 506 (Misc.)
——, with van Thiel, Reuter & Bevere, 1004 (Mal.)
Sautter, V., with Lépine, 728 (Rab.)
Sawitz, W., 716 (Hel.)
——, with Faust, d'Antoni, Odom, Miller, Peres, Thomen, Tobie & Walker, 144 (Hel.)
——, Tobie, J. E. & Katz, G., 830 (Hel.)
Scaffidi, V., 989 (Fev.)
Schalm, L., 934 (Misc.)
Schapiro, M. M., Crosby, B. L. & Sickler, M. M., 146 (Hel.)
Scharff, J. W., (788) (Mal.)
——, with Hackett, Russell & White, 820 (Mal.)
Schenken, J. R. & Moss, E. S., 845 (Hel.)
Schlackman, M., with Boyd, 10 (Mal.)
Schlenova, M. Th., 490 (Mal.)
Schlesinger, A., 922 (Mal.)
Schmidt, H. L., Jr. & Sydenstricker, V. P., 555 (Pel.)
——, with ——, Fulton, New & Geeslin, 551 (Pel.)
Schneider, J., with Decourt, 586 *bis* (Mal.)
——, with Mollaret, 817 (Mal.)
Schneider, M., with Blatt & Hoffman, 196 (Rab.)
Schretzenmayr, A., 445 (Leish.)
——, Chue Sufui & Tsen Hyen-Wun, 445 (Leish.)
——, A. & Lancaster, R. L., 424 (Misc.)
Schroeder, A. G., with Gunther, 1000 (Fev.)
Schüffner, Walch-Sorgdrager & Hoekstra, M., 18, 25 (Y.F.)
Schüffner, W., 634 (Y.F.)
——, Snijders, E. P. & Walch-Sorgdrager, B., 636 (Y.F.)
Schüffner, W. A. P., 129, 401 (Mal.)
——, with Walch-Sorgdrager & Bohlander, 116 (Lept.)
Schütze, H., 971, 974 (Pl.)
Schwartz, S. C., with Simmons, Callender, Curry & Randall, 874 (B.R.)
Schweickhardt, E., 471 (Fev.)
Schwetz, J., 783, 808 (Mal.)
—— & Baumann, H., 777 (Mal.)
—— & Geronnez, E., 777, 778 (Mal.)
Scortecci, G., 570 (Vms.)
Scott, H. H., 791 (B.R.)
Scott, J. A. & Barlow, C. H., 36 (Hel.)
Scott, R. B., 324 (Hel.)
Seal, S. C., with Linton & Mitra, 371 (Chl.)
——, with ——, Shrivastava & Mookerji, 370 (Chl.)
Seal, S. C. & Mitra, B. N., 897 (Chl.)
——, with Read, Singh & Bose, 895 (Chl.)
Sefton, B., 117 (Lept.)
Segal, A., 719 (Hel.)
Seheult, R., (828) (Hel.)
Sehra, K. B., with Iyengar & Mukerji, 855 (Vms.)
Sellards, A. W., with Bennett & Baker, 639 *bis* (Y.F.)
—— & Pinkerton, H., 242 (Lep.)
——, with ——, 242 (Lep.)
Sen, A. K., with Chopra, Pasricha, Goyal & Lal, 909 (Diet.)
Sen, B., with Chopra & Das Gupta, 499 (Mal.)
Sen, P., (136) (Mal.)
Sen, P. N., (348) (Misc.)
Senekji, H. A., 1024 (Leish.)
Senevet, G. & Abonnenc, E., (780) (Mal.)
Sen Gupta, P. C., Rai Chaudhuri, M. N., Chaudhuri, R. N. & Napier, L. E., 543 (Pel.)
——, with Greval & Napier, 1030 (Leish.)
Sen Gupta, P. N., with Maitra & Thant, 366 (Chl.)
Sergent, A., 108, 768 (R.F.)
Sergent, E., 573, 864 *bis* (Vms.)
——, Fourestier, M. & Galliano, E. J., 716 (Hel.)
——, Parrot, L., Donatien, A. & Lestoquard, F., 1026 (Leish.)
Serra, G., 787 (Mal.)
Seyberlich, A., with Genevray & Bruneau, 904 (Chl.)
de Sèze, S., Laplane, L. & Fimbel, M., 253 (Hel.)
Shalom, E., 454 (Leish.)
Shannon, R. C., 639 (Y.F.)
——, Whitman, L. & Franca, M., 27 (Y.F.)
Shapiro, H. A., Sapeika, N. & Finlayson, M. H., 867 (Vms.)
Shapiro, J. M. & Btesh, S., 1031 (Leish.)
Sharp, N. A. D., 941 (Misc.)
Shattuck, G. C., with Bequaert, J. C., Sandground, J. H., Hilferty, M. M. & Clark, S. D., 607 (B.R.)
Sherwood, R. O., with Faget & Palmer, 260 (Mal.)
Shieber, C., 809 (Mal.)
Shillong, 897 (Chl.)
Shimokawa, H. & Inouye, H., (720) (Hel.)
Shionuma, E., Nagai, K. & Maeda, T., 236 (Lep.)
Shipizina, N. K., 581 (Mal.)
Shmelev, K. & Fedorov, V., (964) (Pl.)
Shortt, H. E., 366 (Chl.), 982 (Fev.)
——, Pandit, C. G., & Rao, R. S., 483 (Fav.)
Shrivastava, D. L., with Linton, Seal & Mookerji, 370 (Chl.)
Shukers, C. F., Langston, W. C. & Day, P. L., 424 (Misc.)
Shukry, H., Mahdi, M. A. & El Gholmy, A. A., 912 (Diet.)
Shulov, A., 571 (Vms.)
Shute, P. G. & Badenski, G., 393 (Mal.)
——, with Sinton, 15 (Mal.)
——, with —— & Hutton, 401, 583, 925 (Mal.)
—— & Ungureanu, E., 810 (Mal.)

Sibi, M., with Slatineanu, Balteanu, Nitzulesco & Levit, 545 (Pel.)
Sicault, G. & Messerlin, A., 395, 781 (Mal.)
Sicé, A. & Torrési, F., 663, 664, 669 (Tryp.)
Sickler, M. M., with Schapiro & Crosby, 146 (Hel.)
Siddons, L. B., with Roy & Chandra, 681 (Mal.)
Siegenbeek van Heukelom, A., (814) (Mal.)
—— & Overbeek, J. G., 258 (Mal.)
Sievers, O., 249 (Hel.)
Sikorav, with Villaret, Justin-Besançon & Klotz, 543 (Pel.)
d'Silva, M., with Fernando, Stork & Sinnatamby, 832 (Hel.)
da Silva, O. L., 692 (Lep.)
Silveira, R., with Falla Alvarez & Bernardino, 605 (Dys.)
Silvetti, L. M., 977 (Pl.)
Simeons, A. T. W., 686 (Mal.)
Simmons, J. S., 425 (Misc.)
——, with Callender, G. R., Curry, D. P., Schwartz, S. C. & Randall, R., 874 (B.R.)
Simons, H., 765 (R.F.)
——, with Nicolle, 746 (Tryp.), 933 (Misc.), 984 (Fev.)
Simpson, V. E., 844 (Hel.)
Singh, G., with Read, Seal & Bose, 895 (Chl.)
Sinha, B. C., 911 (Diet.)
Sinitsin, D., with Kessel, 599 (Dys.)
Sinnatamby, G. R., with Fernando, d'Silva, & Stork, 832 (Hel.)
Sinton, J. A., 396 (Mal.), 941 (Misc.)
——, with Christophers, 390 (Mal.)
——, Hutton, E. L. & Shute, P. G., 401, 583, 925 (Mal.)
—— & Shute, P. G., 15 (Mal.)
Sioli, F., Kentenich, A. & Boldt, E., 494 (Mal.)
Sison, A. B. M. & Tolentino, D. G., (36) (Hel.)
Sitanala, J. B., (247) (Lep.)
Sivalingam, V., 681 (Mal.)
Sjöberg, S.-G., 770 (Lept.)
Slătineanu, A., Balteanu, I., Nitulescu, J. & Levit, V., 545 (Pel.)
——, ——, Sibi, M., Nitzulesco, I. & Levit, V., 545 (Pel.)
—— & Potop, I., (469) (Fev.), 549, 550 (Pel.)
Slimon, J. G., 731 (Rab.)
Sloan, N. R., 238 (Lep.)
Slotta, K. H. & Fraenkel-Conrat, H., 855 (Vms.)
Smith, C. N., with Bishopp, 475 (Fev.)
Smith, D. & D'Amour, F. E., 868 (Vms.)
Smith, D. T., with Ruffin, 551 (Pel.)
Smith, H. F., 382 (Chl.)
Smith, H. H., Penna, H. A. & Paoliello, A., 648 (Y.F.)
Smith, J. E., with Julianelle, Sory & Lange, 457 (Oph.)
Smith, R. O. A., McGuire, J. P., Stephens, E. D. & Lahiri, B. N., 729 (Rab.)
Smith, W. H., 346 (Misc.)
Snelling, J. G., 159 (Misc.)
Snijders, E. P., with Schüffner & Walch-Sorgdrager, 636 (Y.F.)
Sociedad Argentina Patología Regional, 875 (B.R.)
Soeiro, A. & Rebelo, A., 488 (Mal.)
Soeparmo, M., (243) (Lep.)
Sokhey, S. S., 729 (Rab.) 976, 977 (Pl.), 1008 (Mal.)
Soldini, M., 748 (Tryp.)
Soliman, M. A., with Ismail, 886 (Bl.)
Soliman, N., 375 (Chl.)
Sollier, R., 506 (Misc.)
van Someren, V. D., 849 *bis* (Hel.)
Soper, F. L., 18, 633 (Y.F.)
Sorel, F. P. J., 18 (Y.F.)
Sorley, E. R. & Currie, J. G., 400 (Mal.)
Sory, R., with Julianelle, Smith & Lange, 457 (Oph.)
Sotolongo, F., with Kouri, Basnuevo & Anido, 599, 605 (Dys.), 626 (Hel.)
Souchard & Nguyen-van-Huong, 102 (Der.)
South African Institute for Medical Research, 196 (Rab.)
Southby, R., with Symes, 740 (Tryp.)
Southwell, T. & Kirschner, A., 155 (Hel.)
de Souza-Araujo, H. C., 241, 245 (Lep.)
——, with Morales Muñoz & Bernal Londoño, 690 (Lep.)
Spacek, M. R. W., 54 (Sp.)
Speight, A., 243, 537 (Lep.)
Spies, T. D., Aring, C. D., Gelperin, J. & Bean, W. B., 553 (Pel.)
——, Bean, W. B. & Stone, R. E., 554 (Pel.)
——, Grant, H. M. & Huff, N. E., 557 (Pel.)
——, Grant, J. M., Stone, R. E. & McLester, J. B., 555 (Pel.)
——, with Vilter & Bean, 554 (Pel.)
——, with Vilter, R. W. & Vilter, S. P., 548 (Pel.)
Spiliopoulos, G., with Choremis, 398 (Mal.)
Stahnke, H. L., with Kent, 865 (Vms.)
Stalder, H., 54 (Sp.)
Starkoff, O., 715 (Hel.)
Starzyk, J., 464 (Fev.)
Stauber, L. A., 1010 (Mal.)
Stauss, H., 683 (Mal.)
Stefanopoulo, G. J., with Roubaud & Colas-Belcour, 638 (Y.F.)
Stephens, E. D., with Smith, McGuire & Lahiri, 729 (Rab.)
Sternberg, E. J., with Brussin, 112, 113 (R.F.)
Stevenson, D. S. & Heggie, J. F., 252 (Hel.)
Stevenson, L. D., 640 (Y.F.)
Stewart, F. H., 956 (Oph.)
Stewart, M. A. & Mackie, D. B., 316 (Pl.)
Stewart, R. A. & Meyer, K. F., 101 (Der.)
Stiles, W. W., with Morrow, Syverton & Berry, 772 (Lept.)
Stone, R. E., with Spies & Bean, 554 (Pel.)
——, with ——, Grant & McLester, 555 (Pel.)
Stork, G. K. B., with Fernando, d'Silva & Sinnatamby, 832 (Hel.)
Strachan, P. D., 533 (Lep.)
Strain, W. H., with Dobriner & Localio, 547 (Pel.)
Stretcu, I., with Ciuca, Balteanu, Alexa, Boeriu, Rugina & Radianov, 11 (Mal.)
Strickland, C., (8), 137, 682 (Mal.)
Strocchi, O., 348 (Misc.)
Strother-Smith, F. F., 403 (Mal.)
Stürup, G. K., with Bernth, 552 (Pel.)
Sun, C. J., with Yao & Wu, 150 (Hel.)

Sussini, M., 962 *bis* (Pl.)
Suwo, M. & Kin, S., 1021 (Lep.)
Suzuki, R., 1019 (Lep.)
Swartzwelder, J. C., 290 (Am.), 659 (Tryp.)
——, with Craig, 294 (Am.)
Sweet, W. C., 808 (Mal.)
Swellengrebel, N. H. & de Buck, A., 90 (B.R.)
Sydenstricker, V. P., with Schmidt, 555 (Pel.)
——, Schmidt, H. L., Jr., Fulton, M. C., New, J. S. & Geeslin, L. E., 551 (Pel.)
Symes, C. B. & Southby, R., 740 (Tryp.)
Syrkine, S. A., with Moshkovski, 932 (Mal.)
Syverton, J. T., with Morrow, Stiles & Berry, 772 (Lept.)

T.

Taborisky, J., 955 (Oph.)
Taddia, L., 141 (Mal.)
Takahasi, H., 48 *bis* (Y. & S.)
Taliaferro, W. H., 214 (Tryp.)
——, with Bloom, 586 (Mal.)
Talice, R. V., 226, (227), 230, 231 (Tryp.)
——, Alambarri, A. & Regules, U., 227 (Tryp.)
——, Loustau, H. L. & Sapriza, J. P., (227) (Tryp.)
——, de Medina, F. & Rial, B., 226 (Tryp.)
——, Regules, U. & Alambarri, A., 226 (Tryp.)
—— & Rial, B., 754 (Tryp.)
——, —— & de Medina, F., 225 (Tryp.)
—— & Terra-Núñez, G., 225 (Tryp.)
Tamler, N., with Tyndel, 550 (Pel.)
Tanaka, I., with Otomo & Koga, 312 (Pl.)
Tang, C. C., 37 (Hel.)
Tanguy, Y., 604 (Dys.), 917 (Mal.)
Tansurat, P., with Daengsvang, 156 (Hel.)
Tartaglia, P., 769 (Lept.)
Tate, P., with Bishop & Thorpe, 587 (Mal.)
——, with James, 339 (Mal.)
Taylor, F. H., (426) (Misc.)
Taylor, J., 368 (Chl.)
—— & Ahuja, M. L., 376 (Chl.)
Tchalaja, L. E., 284 (Am.)
Tchitchibabine, A. & Hoffmann, C., 580 (Mal.)
Teichler, G., 407 (Misc.), 785 (Mal.)
Teixeira, J. de C., with de Azevedo, 568 (Vms.)
Terada, B., with Hiyeda, 717 (Hel.)
Teriteanu, E., with Ciuca, Badenski & Ionescu, 823 (Mal.)
Terra-Núñez, G., with Talice, 225 (Tryp.)
Thant, U., with Maitra & Sen Gupta, 366 (Chl.)
Theodor, O., with Adler, 1025 (Leish.)
——, with —— & Witenberg, 443 (Leish.)
van Thiel, P. H., 489 (Mal.)
——, P. & Bevere, L., 778 (Mal.)
——, with Reuter, J., Sautet, J. & Bevere, L., 1004 (Mal.)
Thiroux, A., 217 (Tryp.)
Thomas, H. & Ting, L., 380 (Chl.)
Thomas, R. C. & Millen, R. M., 888 (Bl.)
Thomen, L. F., with Faust, d'Antoni, Odom, Miller, Peres, Sawitz, Tobie & Walker, 144 (Hel.)
Thompson, T. O., 990 (Fev.)
Thorpe, M. V., with Bishop & Tate, 587 (Mal.)
Tibiriçá, P. Q. T., 29 (Y.F.)
Tibourskaja, N. A., 687 (Mal.)
Tiflov, V. E. & Potapov, V. D., 973 (Pl.)
Tilli, P., 335 (Mal.)
Tillier, H., with Loubeyre & Foissin, 296 (Am.)
Ting, L., with Thomas, 380 (Chl.)
Tisseuil, J., 240, (534), 693, 1019 *ter* (Lep.)
—— & Gilhaumou, F., 694 (Lep.)
—— & Rivoalen, P., 693 (Lep.)
Tivollier, with Dusan & Lombard, (535) (Lep.)
——, with Vigne, Vignoli & Bonnet, 693 (Lep.)
Tjitrohoepojo, with Sardjito & Mochtar, 235 (Lep.)
Tobie, J., with Faust, d'Antoni, Odom, Miller, Peres, Sawitz, Thomen & Walker, 144 (Hel.)
——, with Sawitz & Katz, 830 (Hel.)
Tolentino, D. G., with Sison, (36) (Hel.)
Tolentino, J. G., 531 (Lep.)
Torrealba, J. F., with Dias, 229 (Tryp.)
Torres, C. M. & de Azevedo, A. P., 844 (Hel.)
Torrési, F., with Sicé, 663, 664, 669 (Tryp.)
Tötterman, G., 248 (Hel.)
Toullec, 560 (Bb.)
Toumanoff, C., 391 *bis*, 810 (Mal.)
——, with Genevray & Hoang-Tich-Try, 131 (Mal.)
Toyoda, K., with Yoshida, 722 (Hel.)
Trabaud, J. & Rabïe, D., 300 (Am.)
Trapani-Angileri, R., 124 (Bl.)
Trapesontzewa, C., 956 (Oph.)
Traut, E. F. & Logan, C. E., 157 (Misc.)
Travassos, J., 478 (Fev.)
Trengganu, 173 (Rep.)
Trensz, F., 394 (Mal.)
Trinquier, E., with Reynes, 667 (Tryp.)
Trolli, G., 501 (Misc.), 912 (Diet.)
Trotter, F. E., 973 (Pl.)
Trowell, H. C., 180, 876 (B.R.), 938 (Misc.)
Truche, C., with Nicolau, Cruveilhier, Kopciowska & Viala, 195 (Rab.)
Trumpe, D., with Kettelkamp & Murphy, 347 (Misc.)
Tschan, T. J., with Otto & Au, 41 (Hel.)
Tschilow, K., 784 (Mal.)
Tsen Hyen-Wun, with Schretzenmayr & Chue Sufui, 445 (Leish.)
Tsuchiya, H., 285 (Am.)
Ts'un, T. & Chung, H.-L., 111 (R.F.)
Tudhope, G. R., with Rogers, 253 (Hel.)
Tumansky, V., 964 (Pl.)
T'ung, T., with Chung, 715 (Hel.)
Tupa, A. & Ciuca, M., with Badenski, G. & Ionescu, P., 822 (Mal.)
Turner, T. B., with Ferris, 45, 47 (Y. & S.)
Tyndel, M. & Tamler, N., 550 (Pel.)
Tyzzer, E. E. & Geiman, Q. M., 598 (Dys.)

U.

Uganda Protectorate, 63 (Rep.)
Ugarriza, R. G., 696 (Lep.)
Underwood, P. G. & Harwood, P. D., 840 (Hel.)

Ungo-Mugdan, A., 931 (Mal.)
Ungureanu, E., with Shute, 810 (Mal.)
Uttley, K. H., 307 (Pl.)

V.

Valaoras, V. G., with Livadas & Canellakis, 397 (Mal.)
Valcke, G. & Van den Berghe, L., 886 (Bl.)
Valette, G. & Rollé, R., 304 (Am.)
Valleteau de Mouillac, Duliscouet & Delacoux des Roseaux, 450 (Leish.)
Van den Berghe, L., 591 (Hel.), 634 *bis*, 641, 642 (Y.F.)
—— & Denecke, K., 628 (Hel.)
——, with Valcke, 886 (Bl.)
Van den Branden, F., 668, 743, 744 *bis* (Tryp.)
—— & Pottier, R., 671 (Tryp.)
Van der Walle, N., 117 (Lept.)
Vanni, V., 453 (Leish.), 724 (Hel.), 1034 (Leish.)
Vardon, A. C. & Datta Roy, B. K., 377 (Chl.)
Varela, G., 865 (Vms.)
—— & Sánchez Posada, E., 866 (Vms.)
Varela Zequeira, R., 416 (Misc.)
Vargas, L., 779 (Mal.)
Vassiliadis, P. C., 27 (Y.F.)
Vaucel, M., 114 (Lept.)
Vaughan, J., 422 (Misc.)
Vazquez-Colet, A. & Africa, C. M., 42 (Hel.)
Vecchio, F., with Murano, 1029 (Leish.)
van Veen, A. G., 415 (Misc.)
Veeraraghavan, N. & Philipsz, G. L. C., 193 (Rab.)
Veintemillas, F., 1002 (Fev.)
Velds, M., 694, (697) (Lep.)
Vellard, J., 856, 858, 859 (Vms.)
Verbunt, J. A., with Boedyn, 103 (Der.)
Versiani, W., with Martins, 39, 623 (Hel.)
Verslagen en Mededeelingen Betreffende de Volksgezondheid, 401 (Mal.)
Viala, C., with Cruveilhier, 197 (Rab.)
—— with —— & Dieryck, 732 (Rab.)
——, with —— & Lépine, 197 (Rab.)
——, with Nicolau, Cruveilhier, Truche & Kopciowska, 195 (Rab.)
Vidal, A., 914 (Diet.)
Vierthaler, R. W. & Boselli, A., 742 (Tryp.)
Vigne, P., 452 (Leish.)
——, Vignoli, Bonnet, R. & Tivollier, 693 (Lep.)
Vignoli, with Vigne, Bonnet & Tivollier, 693 (Lep.)
Viljoen, N. F., 254 (Hel.)
Villain, with Durand, 932 (Mal.)
Villaret, M., Justin-Besançon, L., Klotz, H. P. & Sikorav, 543 (Pel.)
Villela, G. G., (247), 696 (Lep.)
Vilter, R. W., Vilter, S. P. & Spies, T. D., 548 (Pel.)
Vilter, S. P., Bean, W. B. & Spies, T. D., 554 (Pel.)
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